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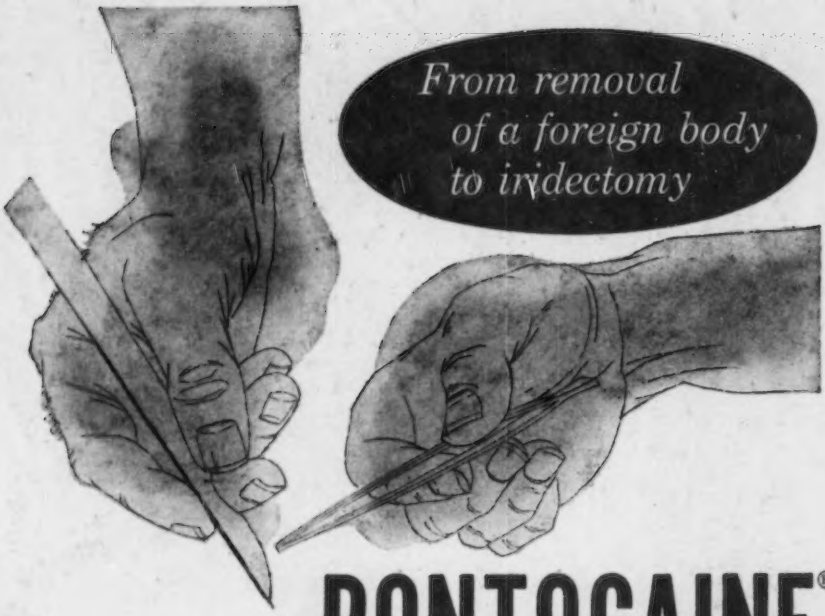
ANNUAL MEETING OCTOBER 12-17, 1952, CHICAGO



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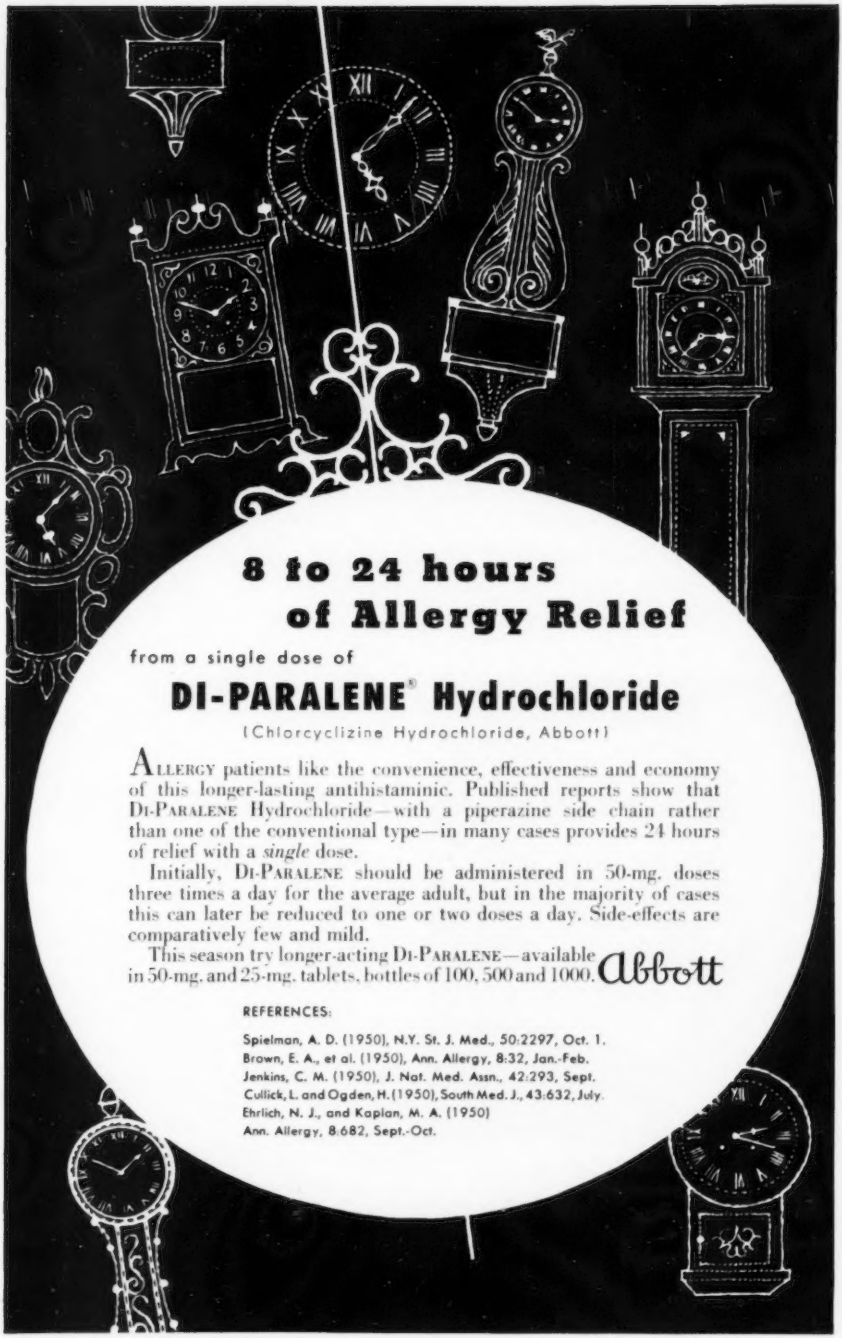

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REFERENCES:

Spielman, A. D. (1950), N.Y. St. J. Med., 50:2297, Oct. 1.
Brown, E. A., et al. (1950), Ann. Allergy, 8:32, Jan.-Feb.
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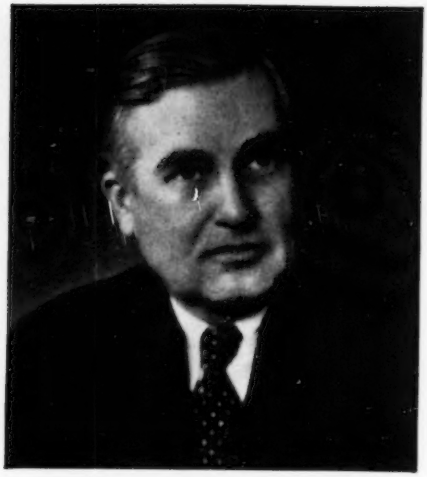
Ehrlich, N. J., and Kaplan, M. A. (1950)
Ann. Allergy, 8:682, Sept.-Oct.

MRS. ELEANOR ROOSEVELT

World-famous wife and mother; Senior United States Representative of the United Nations General Assembly; author, radio and television commentator; internationally respected and admired for her interest in, and understanding of, all peoples.



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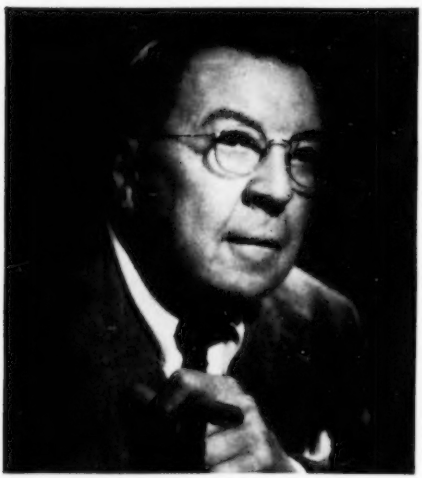

MR. RUPERT HUGHES

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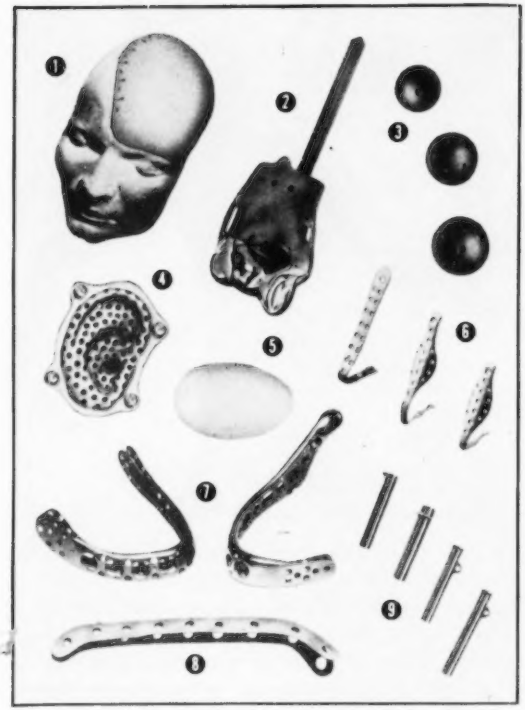

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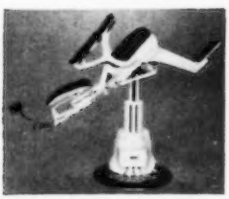

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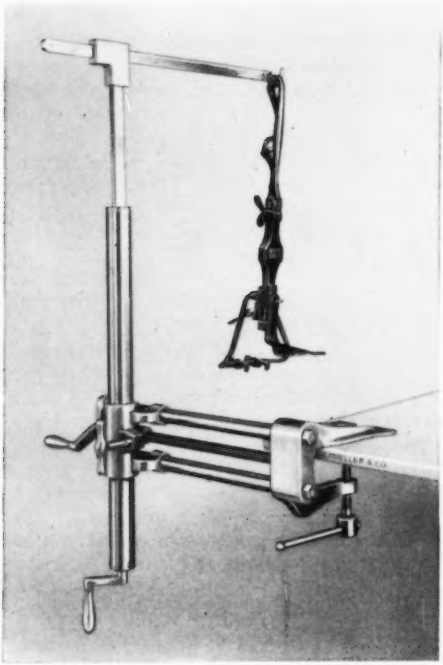

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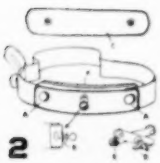

2

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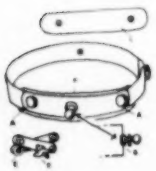

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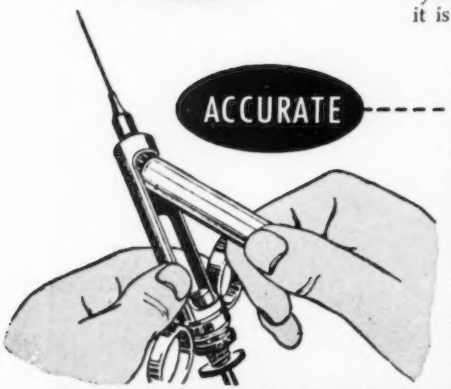

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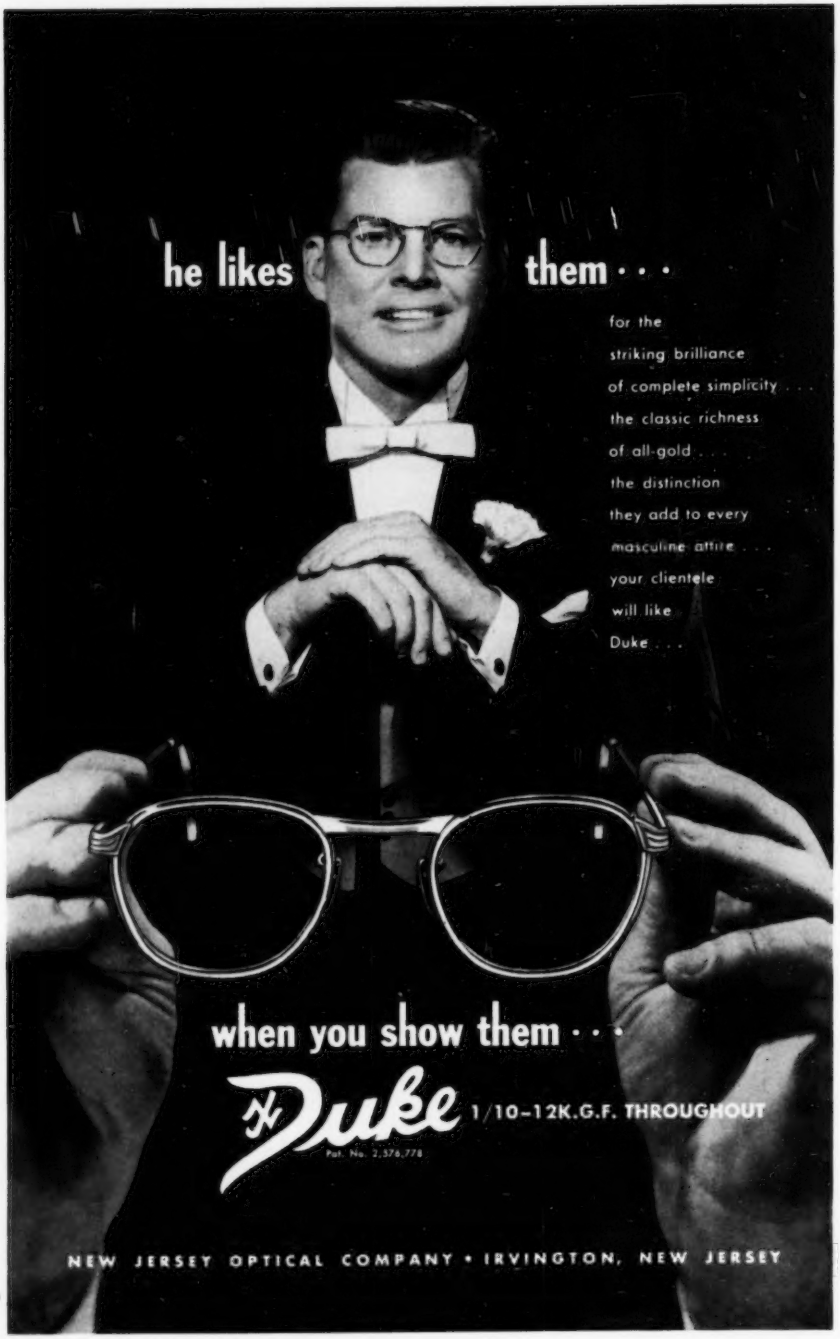
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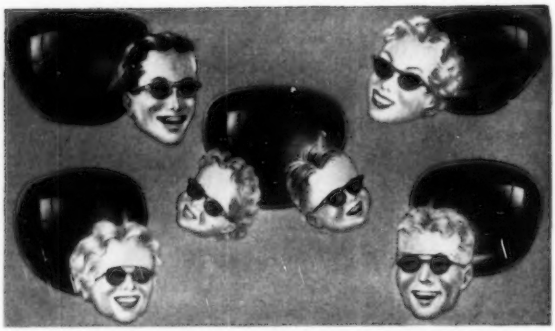

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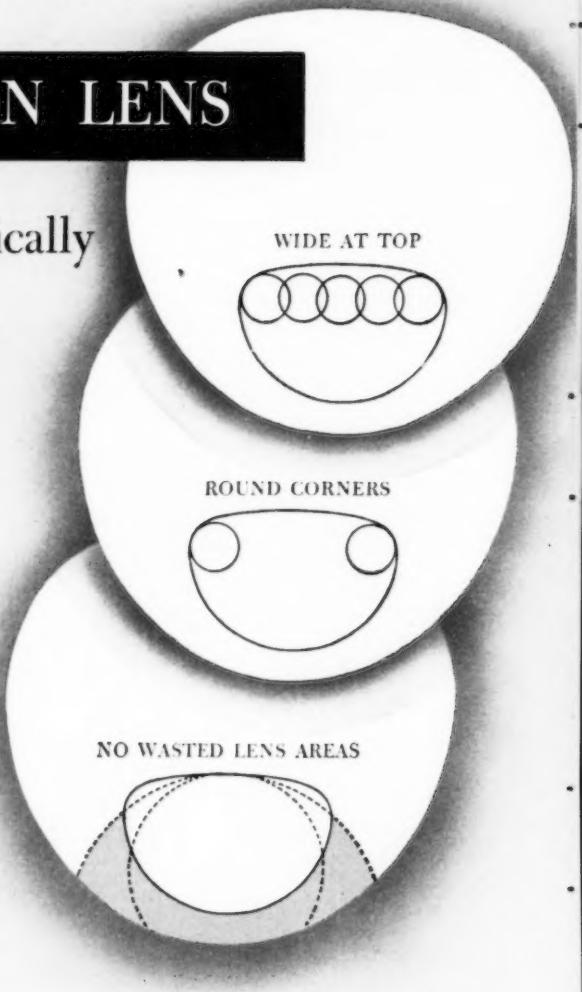

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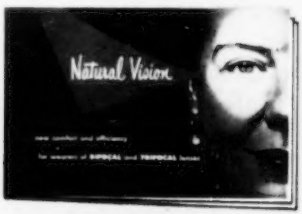

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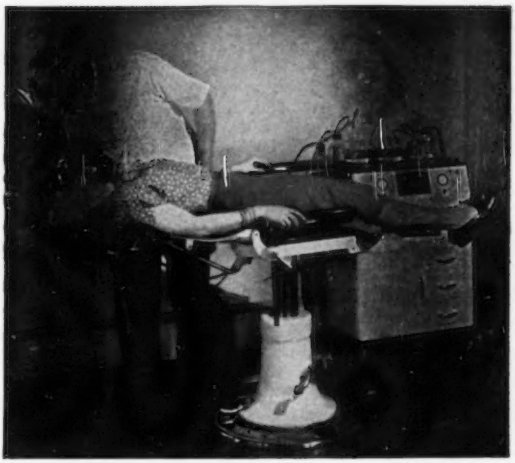

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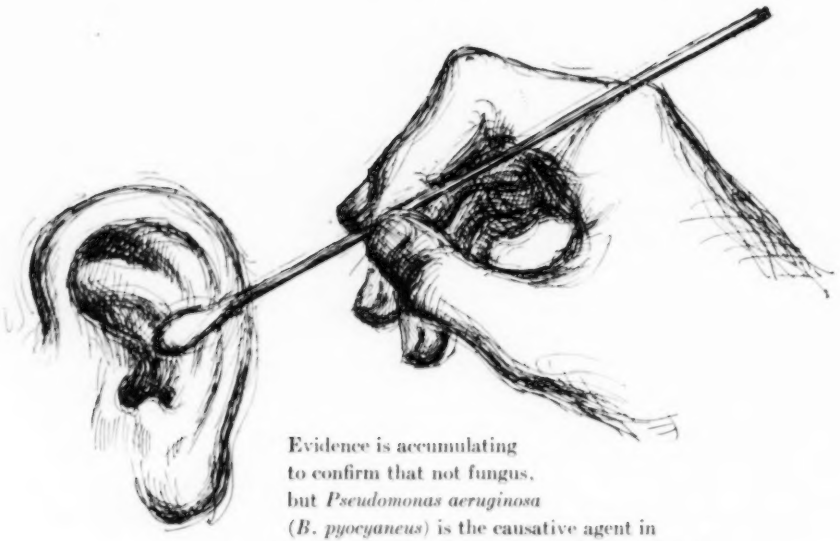

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1. Senturia, B. H.: Laryngoscope, 55:277, 1945.
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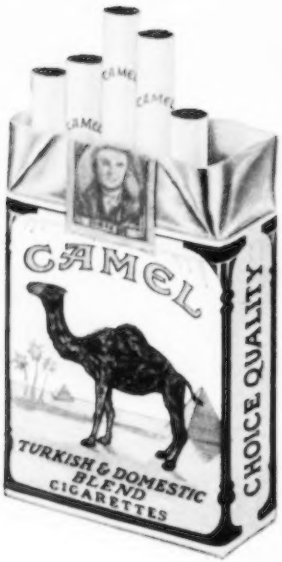

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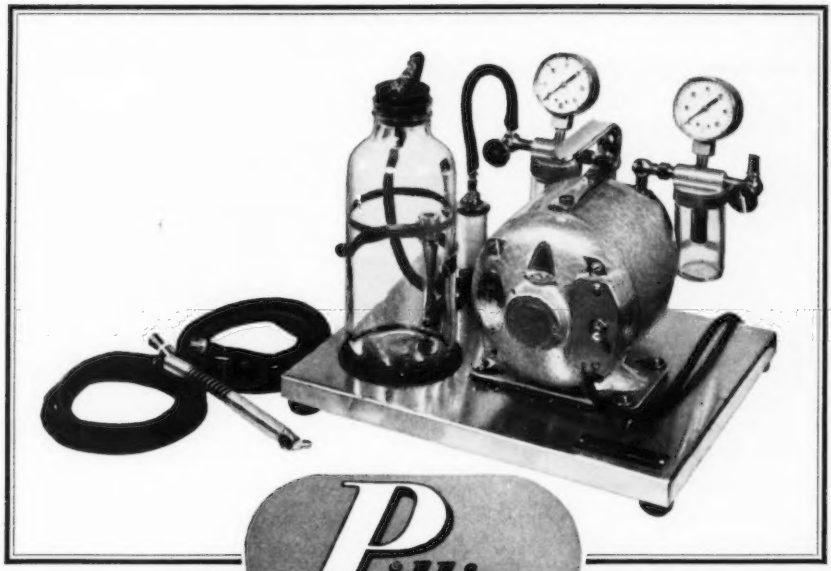

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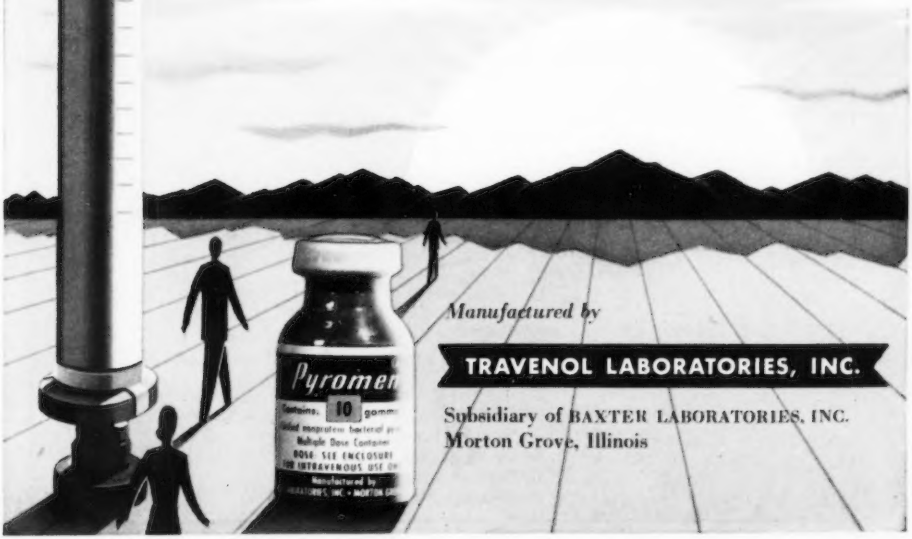

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INTRODUCTION AND DEFINITION OF TERMS

LAWRENCE T. POST, M.D.
ST. LOUIS, MO.

THE Academy is presenting this symposium on primary retinal detachments and those following direct or nonperforating injury to the eye principally in the hope that from this study will evolve improvements in procedures for the diagnosis, prognosis, and treatment of this condition. It will be our purpose to confine the discussion to essentials and to present them, in so far as possible, in such simple form as to be of practical value to those who infrequently operate on detachments. Because of time limitation, individual references have been largely omitted.

It is our opinion that breaks in the continuity of the retina are the most essential factor in the production of retinal detachment and that the following may be causes for such breaks: (1) a localized atrophying process in the retina, (2) a localized vitreous pull, and (3) trauma.

The term "break" (short for break in the continuity of the retina) will be used to indicate holes, tears, or disinsertions. It was agreed that the Committee would discuss the so-called primary detachment as well as those following direct nonperforating injuries and indirect trauma, but would not discuss detachments associated with perforating injuries, tumors, and massive exudates.

By retinal break is meant any break in the continuity of the retina. By retinal tear is meant a break created by vitreous traction and characterized by the presence of a retinal flap or operculum. A retinal hole is a round or oval opening occasioned by extreme thinning of the retina. Retinal dialysis and retinal disinsertion are used synonymously and refer to a break in the retina occurring at its peripheral insertion. Demarcation line is used synonymously with friction line and by this is meant a pigmented or white line or band located in the choroid and retina. It often forms at the limit between the detached and attached parts of the retina when this limit remains for about three months or more in the same location.

An enormous volume of literature has accumulated in the last thirty-five years on the subject of retinal detachment, the stimulus for this having been the classical work of Gonin. Since it is impracticable to review here this voluminous mass or to evaluate all of it and since a complete discussion of the subject would occupy endless days, the essayists have had to confine themselves to somewhat dogmatic statements, thus possibly over-simplifying the subject, fully recognizing, however, that some of the material is really controversial.

The symposium is intended to express the consensus of the group participating in it, and where an individual promulgates an idea contrary to the general opinion, he will make this clear in his remarks.

Analyses can be based on anatomic reattachments or on visual results. For purposes of statistical analysis, the

Presented at the Fifty-Sixth Annual Session of the American Academy of Ophthalmology and Otolaryngology, Oct. 14-19, 1951, Chicago, Ill.

Committee has selected the anatomic viewpoint and classified complete reattachment of the retina for a period of not less than six months as a cure. Obviously such a time limit is arbitrary, but some definite period had to be chosen for the statistical analyses. The doctors participating in the symposium are agreed that the percentage of spontaneous detachments occurring six months or longer after successful surgery is very small.

There will be some discussion and classification of degrees of failure. Among the cases considered failures according to this definition, there will be some patients who have derived considerable functional benefit from the operation. Conversely, there are cures under our arbitrary definition that have not gained visually or may even have lost vision.

Symposium: Retinal Detachment

* * *

ETIOLOGY AND PATHOLOGY

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THIS discussion will deal for the most part with the pathogenesis and sequence of events in the formation of retinal detachment. The actual morbid anatomy will be shown, but always with the idea of trying to answer the question of why the detachment occurred, and what is its clinical significance. It will be my purpose to try to explain the clinical picture of detachment of the retina from the pathologic findings.

I have divided the discussion into three parts:

1. Anatomic changes
2. Sequence of events
3. Microscopic changes following treatment

FIG. 1.—Detachment of the retina of long standing. This shows a break in the retina near the periphery at "A."

ANATOMIC CHANGES

The pathologic findings in eyes removed with a primary detachment of the retina are well known, and many of these changes have a direct bearing on the success of treatment. A break in the continuity of the retina is generally considered an all important factor in the development of the separation of the retina.

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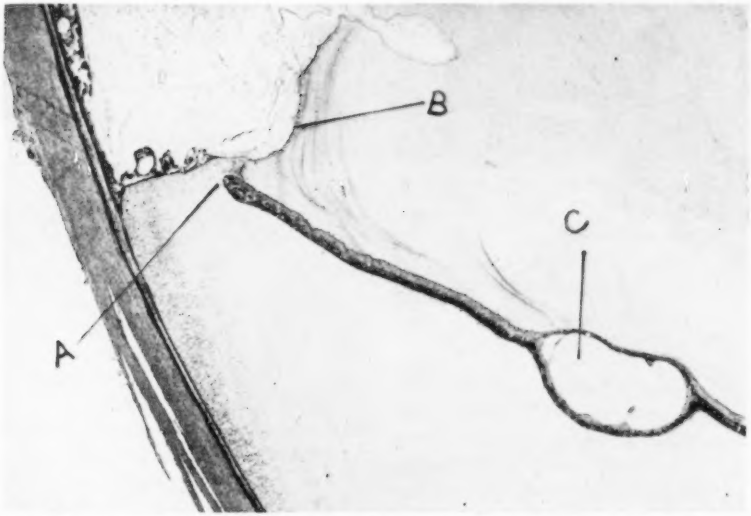

FIG. 2--High power of area indicated in figure 1. (A) Retinal break with smooth sealed edges; (B) Dense vitreous strand attached to shrunken contracted vitreous; (C) Large cystic area in retina following prolonged detachment.

Figure 1 shows a detachment of the retina of long standing. There is a break in the retina near the ora as indicated by the arrow. Figure 2 is the area indicated as "A" in figure 1. Here one can see a break in the retina (A), the edges of which are rounded and smooth indicating that this break existed in vivo. Attached to the peripheral lip of the break is the contracted vitreous (B). Following the occurrence of the break, fluid has migrated beneath the retina and caused a separation. If the detachment exists for many months, cystic changes may develop, as can be seen in the section shown at (C).

With the retina detached for a long period of time, the entire outer portion of the retina may become cystic. These cystic changes will render the hope of restoring vision a rather dim one. Figure 3 shows very extensive cystic changes in the retina with a detachment of long standing.

It is interesting to note that these cysts nearly always protrude into the subretinal space and thereby leave the inner surface of the retina relatively smooth. Generally these cystic changes begin in the outer layers of the retina, and they are most probably due to the lack of nutrition in the outer layers which are normally supplied by the choriocapillaris of the choroid.

FIG. 3—Detachment of long standing with marked cystic degeneration. These cysts extend into the subretinal space and generally involve the outer layers of the retina.

FIG. 4.—Detachment of long standing with gliosis retinae.

Cystic changes may not be the only degenerative change to decrease the chance for a favorable outcome when attempts are made to reattach the retina. With a detachment of long standing the nuclear layers may decrease and fuse to form one layer and the ganglion cells tend to disappear. The rods and cones also become sparse. There remains only the glial supporting tissue. In time the glial supporting tissue contracts and shortens the retina, making for a greater difficulty in reattachment.

Figure 4 shows a detachment of long standing, and figure 5 is a high power view of the area indicated in figure 4. Figure 5 demonstrates the fusion of the two nuclear layers into a single layer. There is a loss of ganglion cells, and on the outer surface of the retina only remnants of the rods and cones can be seen. There is some dispersion of pigment along the outer surface of the retina.

In addition to gliosis, shrinkage may take place because of other changes. This has been explained on the theory that a proliferation of the nonpigmented epithelial cells of the ciliary body extend over the surface of the retina and later contract. It has also been advanced that cells normally present on the inner surface of the retina proliferate and contract. Whatever the origin of these cells, there is often evidence of their contracture drawing the retina into folds.

Figure 6 shows a detachment of long standing with marked contracture of the retina and a definite membrane on

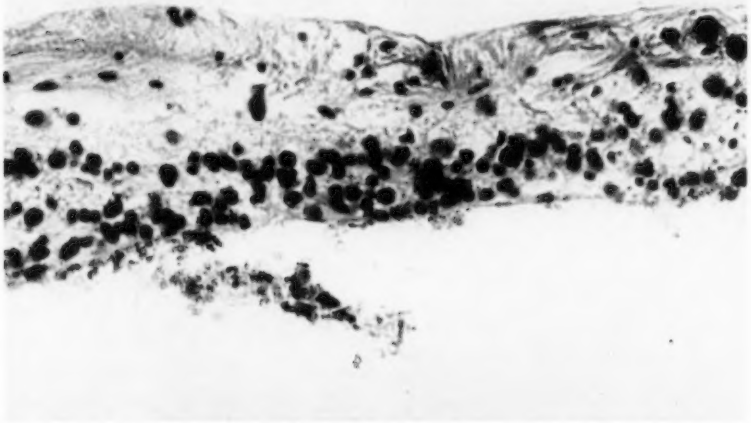

FIG. 5.—High power of area indicated in figure 4. There is a loss of nuclear elements in the retina and a complete absence of ganglion cells. In the outer surface of the retina there are some fragments of rods and cones as well as a dispersion of pigment from the pigment epithelial cells.

FIG. 6.—A detachment of long standing with contracture of the retina due to a proliferation of a cellular membrane on the inner surface of the retina.

the inner surface. The contracture of this membrane has thrown the retina into folds and scallops. The retina has been so shortened by this process that it would not be possible to reattach it by the usual diathermy procedure. Figure 7 shows a high power of the area indicated in figure 6. One can see a definite cellular membrane on the inner surface of the retina, the contracture of which has caused the retina to be thrown into folds and scallops.

For some time the detachment of the retina may remain localized in one area. The outer limits of the detachment may be characterized clinically by a greyish or pigmented line. The subretinal fluid exerts a pull on the retina when the eye is set into motion. Such a force has a tendency to separate the retina from the choroid, but if this force is not too strong it acts as an irritant and causes a dense chorioretinal adhesion. A continual to-and-fro motion of the retina due to the momentum of the subretinal fluid is a continual irritant at the point of attachment of the retina to the choroid. Here there may be a proliferation of the

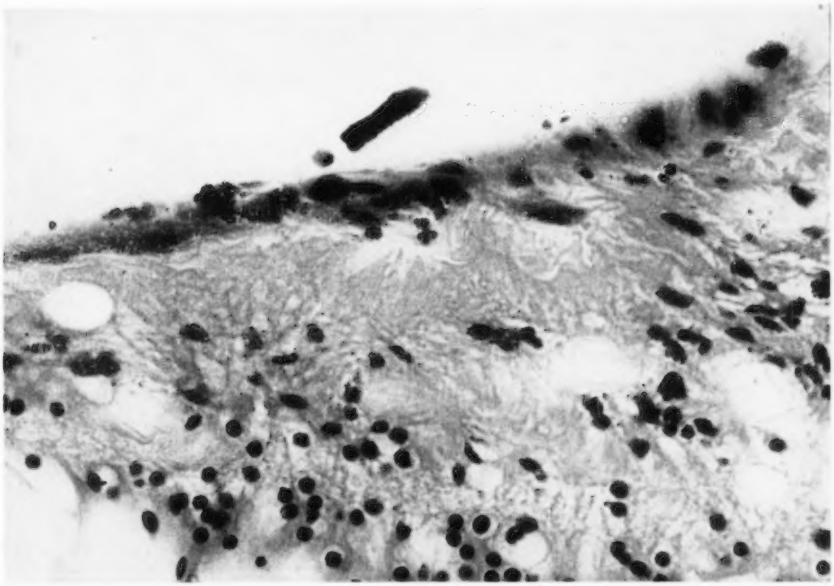

FIG. 7.—A high power of the area indicated in figure 6 showing the cellular membrane on the inner surface of the retina. A contracture of this membrane has caused a shrinkage of the retina with some disorganization of the retinal elements.

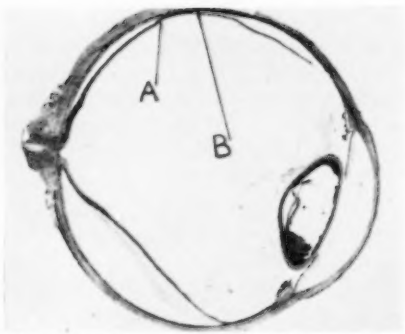

FIG. 8.—Detachment of the retina of long standing with friction lines at points "A" and "B."

pigment epithelium and a wearing away of the rods and cones (area "A" in fig. 8). A dense adhesion is finally formed between the choroid and retina at this point. Degenerative changes take place, as can be seen by the numerous cysts in the nuclear layer. A proliferation and metaplasia of the pigment epithelium may later contract and pull the retina into folds at this point. This metaplasia of the pigment epithelium causes a dense chorioretinal adhesion (fig. 9). Figure 10 (area "B" in figure 8) shows a metaplasia of the pigment epithelium causing a dense chorioretinal adhesion. These so-called friction lines may degenerate and contain calcium (fig. 11). Three things can happen at these friction lines: (1) the adhesion may be sufficiently dense to localize the area of detachment, or (2) there may be sufficient force exerted by the subretinal fluid to strip the retina further causing a greater area of detachment, or (3) the retina may tear at this point of adhesion, thereby forming a secondary break. This will be discussed later.

The pigment epithelium has a tendency to proliferate following any irritating factor, whether it be from diathermy or from toxic products of the

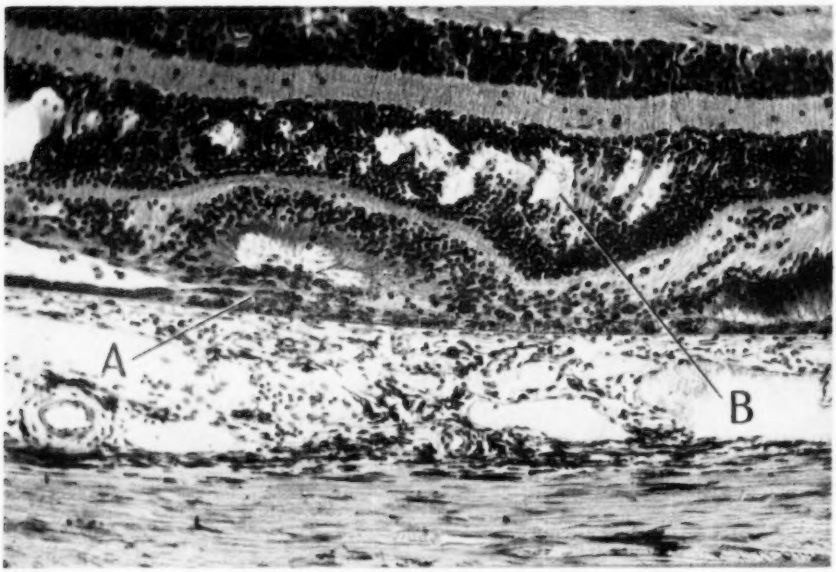

FIG. 9.—High power of area indicated as "A" in figure 8. A chorioretinal adhesion due to the irritation factor of the continual motion and pull at an edge of the detachment. (A) Metaplasia of the pigment epithelium, the contracture of which has caused a distortion of the retinal elements. (B) Cystic changes in the outer nuclear layer. Notice the loss of rods and cones in the friction line.

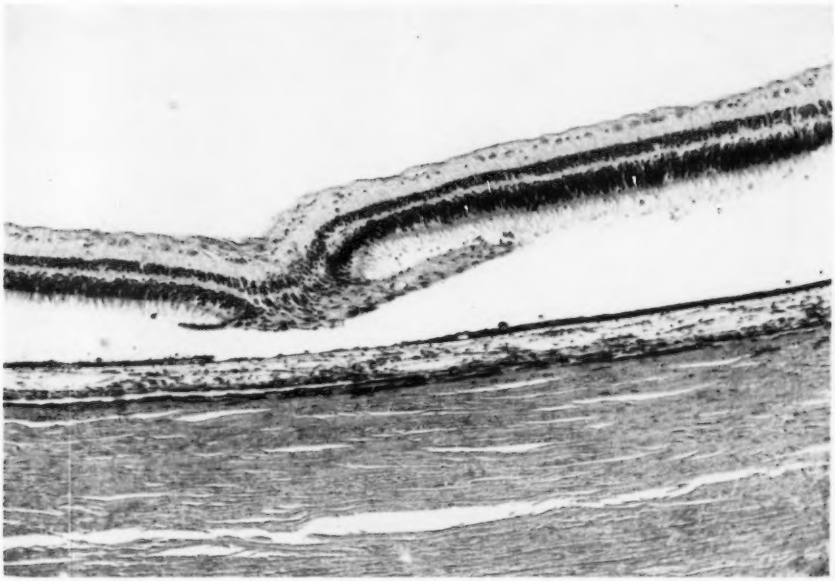

FIG. 10—Area "B" indicated in figure 8, showing metaplasia of the pigment epithelium causing a dense chorioretinal adhesion. The detachment of this area is an artefact due to contracture at fixation.

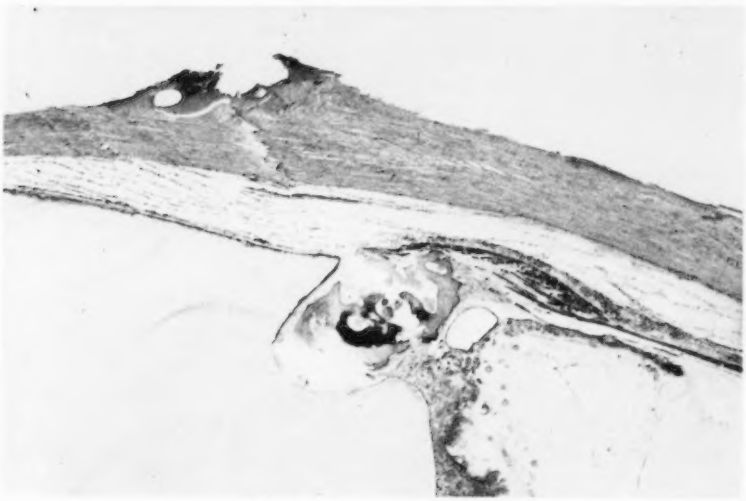

FIG. 11—Friction line with calcium.

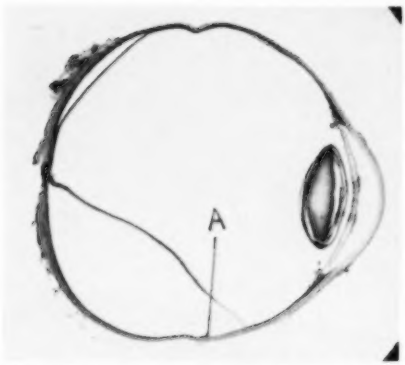

FIG. 12.—A detachment of the retina of long standing with proliferation of pigment epithelium.

degenerating retina. Figure 12 illustrates a detachment of the retina with proliferation of the pigment epithelium at area "A." Figure 13 is the high power of area "A" in figure 12, clearly showing the proliferation of the pigment epithelium.

Many other pathologic changes are found in the globes with detachment of the retina but space will not permit a full discussion of these many changes in this presentation.

SEQUENCE OF EVENTS

In the study of pathologic specimens it is often possible to determine the pathologic processes in their chronologic order. By piecing together these isolated facts we are often able to get the usual sequence of events that take place in the formation of detachment of the retina. It has been thought generally that the vitreous plays a positive role in the formation of detachments of the retina. Processes may take place to cause the vitreous to become detached posteriorly and begin to shrink. The space formed by the shrunken vitreous body is filled with a fluid similar to aqueous. This is difficult to prove pathologically, but it has been my experience to see such contracture in the globes opened for study. One does not see such changes in young normal eyes removed and studied. Clinically, a detachment of the vitreous can be seen with the slit lamp. Figure 14 shows a posterior detachment and shrinkage of the vitreous in a globe opened after enucleation.

Motion of the eye may cause the fluid between the retina and the vitreous to exert sufficient force to produce a fur-

FIG. 13.—High power of area indicated in figure 12 showing a proliferation of the pigment epithelium following a detachment of the retina of long standing.

FIG. 14—Posterior detachment and shrinkage of vitreous.

ther detachment of the vitreous body. If there are pre-existing retinovitreous adhesions, such a force, set in motion by the rotary action of the globe, may tear the retina and cause a break in the continuity. Once the retina has been torn, fluid may then migrate into the subretinal space and cause a separation.

Figure 15 shows a detachment of the retina following a subretinal hemorrhage. There is also a detachment of the

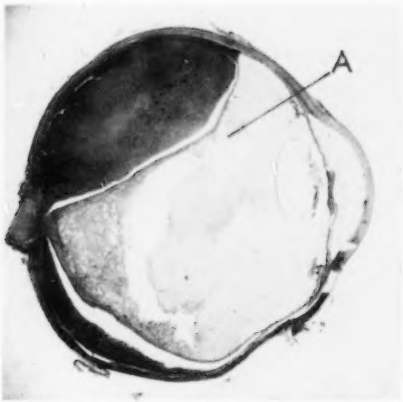

FIG. 15—A detachment of the retina following a subretinal hemorrhage.

vitreous. In figure 16, a high power of the area indicated in figure 15, one can see the detachment of the vitreous, which has become shrunken and separated from the retina.

In addition to a detachment and shrinkage of the vitreous, certain degenerative changes take place. The lique-

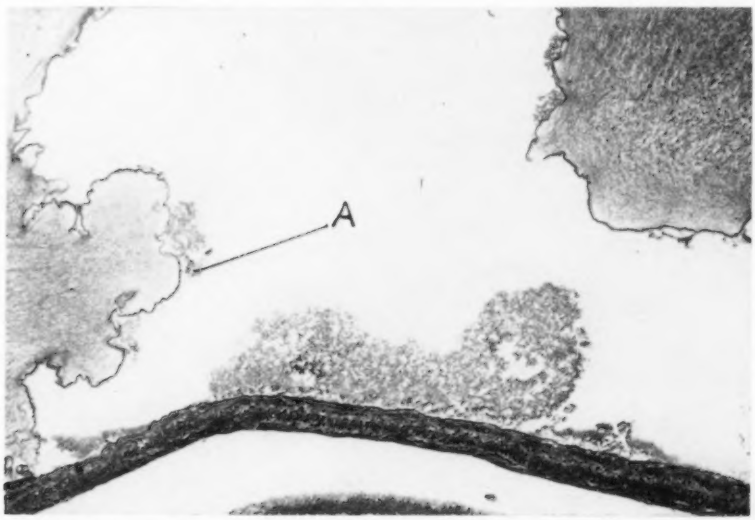

FIG. 16—High power of area "A" in figure 15. A detachment and shrinkage of the vitreous demonstrating the contracted vitreous following a detachment of the retina. (A) Shows the condensation of the vitreous (chromic acid fixative).

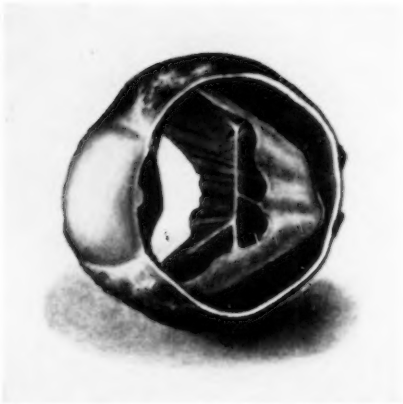

FIG. 17.—Artist's drawing of a detachment of the retina with vitreous strands. This shows the force and relationship of these strands to the vitreous break as seen above.

faction and condensation may be in the form of strands. These strands may contract and exert force at a localized area on the retina and finally cause a break. An artist's drawing (fig. 17) shows the direct relationship of the force of these vitreous strands in the presence of a retinal break.

In conjunction with the vitreous adhesions and vitreous strands there may be atrophic areas in the retina. Motion of the eye with repeated tugs on these weakened areas may cause the retina to give way and form a break with a subsequent detachment.

Figure 18 shows an atrophic area in the retina with the shrunken vitreous body adherent at this point. Repeated tugs caused by the momentum of the shrunken vitreous body can conceivably cause this area to break and result in a detachment.

To illustrate the above process more clearly the next figures show a detachment of long standing with a break near the periphery (figs. 19 and 20). The high power view of the area indicated in the low power picture of the entire globe shows a break in the retina. The edges of the break are smooth and show some contracture of the inner border of the retina. It is obvious that this break existed in vivo. The same area is shown in figure 21 but at a slightly different level. Here we begin to see the true

FIG. 18.—An atrophic area in retina. The contracted vitreous is adherent to the atrophic area.

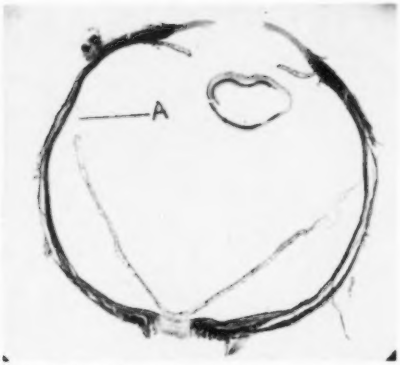

FIG. 19—Detachment of retina with break at periphery "A."

process that existed at the time that the break was formed. At "A" there is a dense vitreous strand adherent to the peripheral lip of the break. This can be followed completely across the globe. At "B" the edge of the break shows cellular infiltration and a definite thinning of the retina.

The condition that probably existed prior to the formation of the break is shown in figure 22. Here we see the dense vitreous strand that extends

FIG. 20—High power of area indicated in figure 19 showing the retinal break.

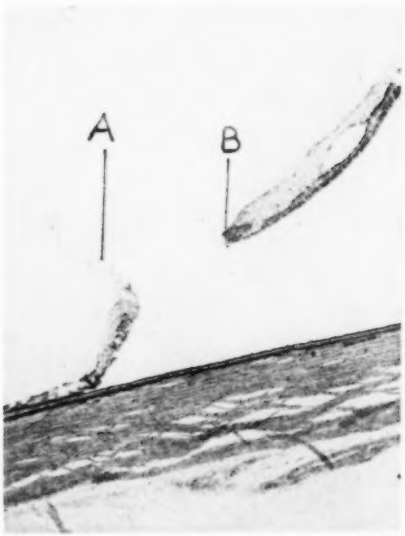

FIG. 21—Same area as figure 20 at a slightly different level. (A) Dense vitreous strand; (B) thinning of retina at edge of break.

across the globe and the thin, atrophic retina adjacent to this area. The normal elements of the retina cannot be distinguished in this atrophic area and it is obviously a weakened portion. Repeated tugs on this dense vitreous strand could easily tear the retina at this point.

This same process can take place in the macula so long as the vitreous is adherent at this point (fig. 23). A high power view of this eyeball clearly shows the break in the macula and the dense vitreous strand adherent to the inner surface of the retina (fig. 24). The contracture of this tissue has caused the retina to fold on itself.

In an old detachment where the vitreous has exerted traction on the retina, degenerative changes take place. Figure 25 shows a detachment of the retina with a dense vitreous strand extending across the inner portion of the eye. The high power of area "A" indicated in figure 25, demonstrates how the repeated tugs on the retina have caused

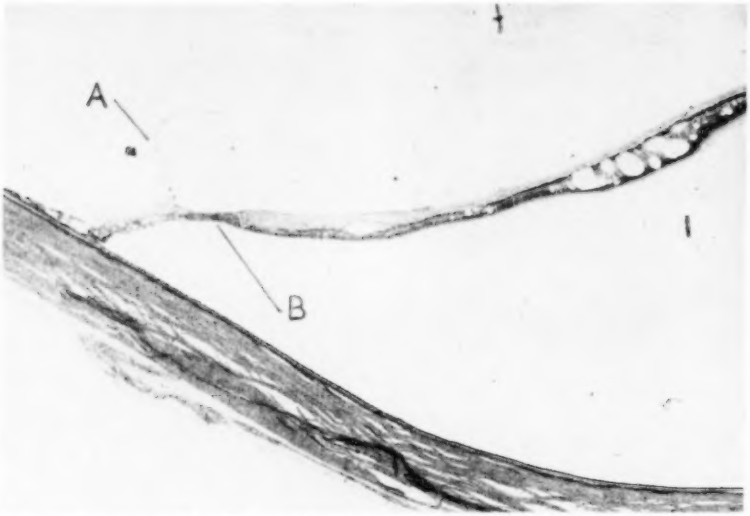

FIG. 22—Same area as figure 20 at different level. (A) Dense vitreous strand; (B) thinning atrophic area of retina near the break.

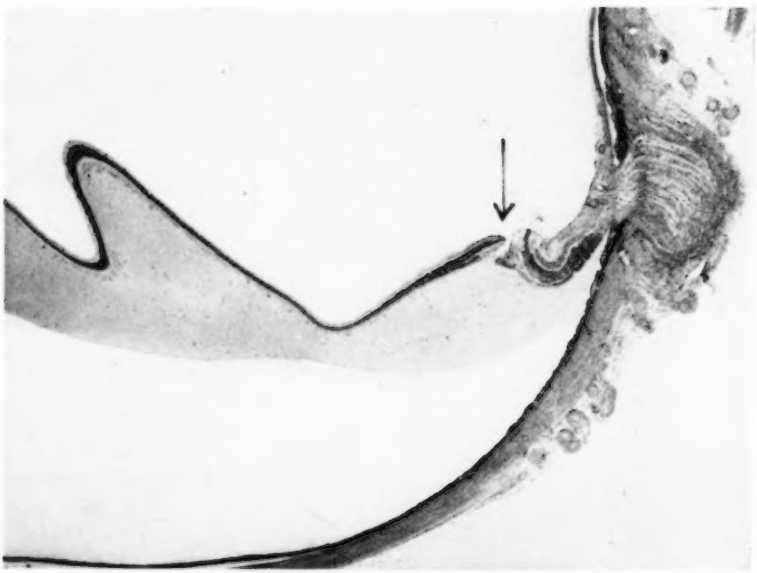

FIG. 23—Detachment of retina with break in macular region.

FIG. 24—A detachment of the retina with a break in the macular region. A vitreous strand is adherent to the retina.

sufficient irritation to manifest some cellular infiltration and degenerative changes of the retina at the point of attachment of this dense vitreous strand (fig. 26). In this particular case the

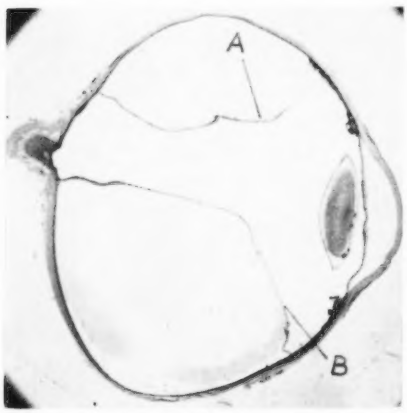

FIG. 25—Detachment with a dense vitreous strand extending across the inner portion of the globe.

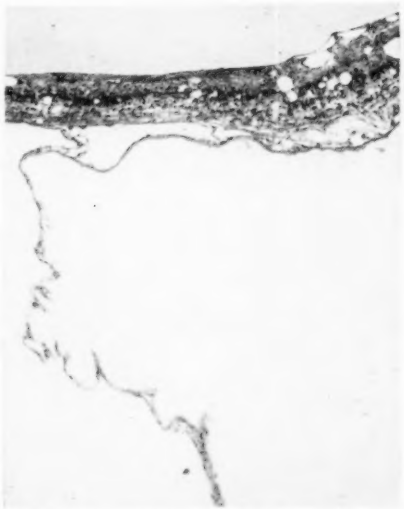

FIG. 26—Area "A" as indicated in figure 25 showing the irritative changes in the retina as a result of repeated traction on the retina at this point.

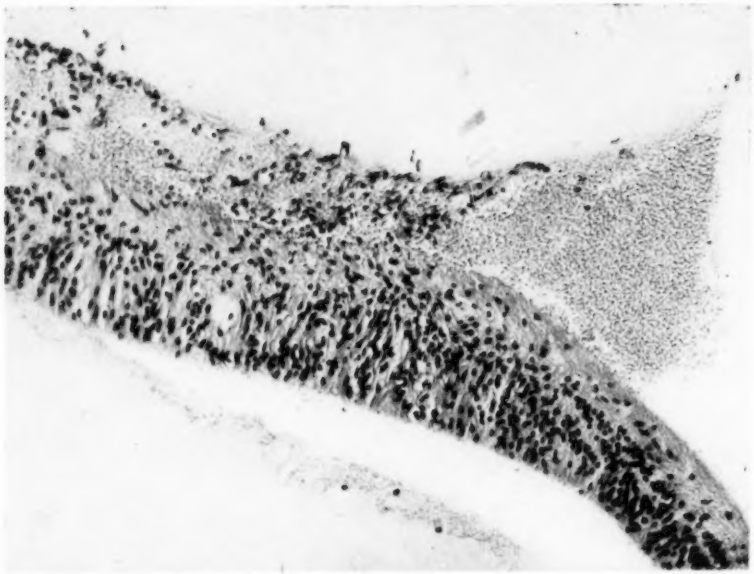

FIG. 27.—A small hemorrhage in the substance of the retina caused by a rupture of a blood vessel due to the traction of the vitreous strand at point "B" as indicated in figure 25.

vitreous strand contains cellular elements and could very easily exert considerable force.

FIG. 28.—Detachment with portion of the retina torn away following contracture of the vitreous.

At the opposite attachment of this dense vitreous strand, area "B" in figure 25, the repeated tugs on this strand have caused the rupture of a small blood vessel and there has resulted a hemorrhage in the substance of the retina (fig. 27). These small hemorrhages no doubt are a frequent occurrence at a point of rupture in the retina. Small hemorrhages such as seen in this specimen may explain the cause for the shower of opacities frequently found in recent detachments.

When the retina gives way by the pull of the vitreous, a piece of the retina may come off with the vitreous and thereby form a complete hole. A small plug or operculum of the retina retracts with the vitreous (fig. 28). An enlargement of the area indicated in figure 28 shows more plainly a small plug of the retina that has been pulled away by the contracture of the dense vitreous strand (fig. 29). One can see that this strand

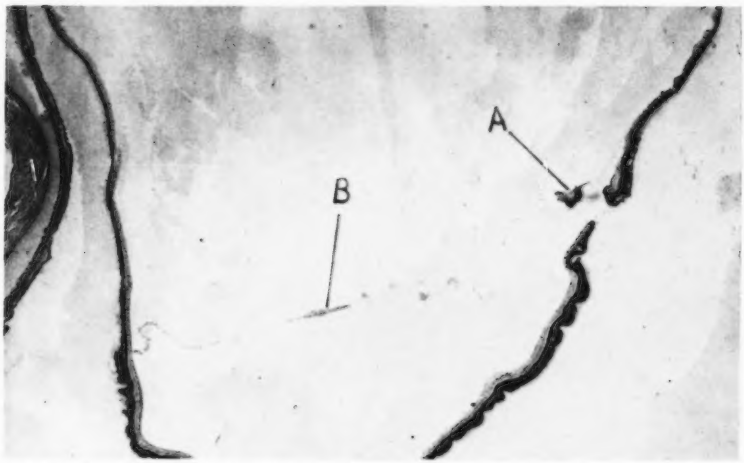

FIG. 29—Detachment with retinal plug pulled into the vitreous cavity by dense vitreous strand. Notice that vitreous strand extends across the globe. (A) Retina pulled into vitreous space; (B) dense vitreous strand.

FIG. 30—High power of "A" in figure 29. Retinal plug pulled in by contracting vitreous.

extends completely across the globe. Figure 30 is a view of still higher power showing the through-and-through break of the retina and the small retinal plug that pulled away and retracted into the vitreous space.

No doubt the peripheral cysts of the retina are a frequent cause for breaks. How often these small cysts cause a detachment of the retina is a debatable

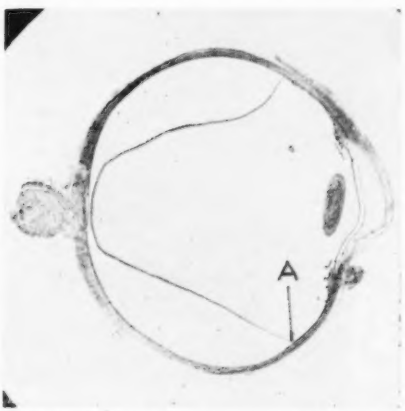

FIG. 31—A globe with peripheral cystic degeneration.

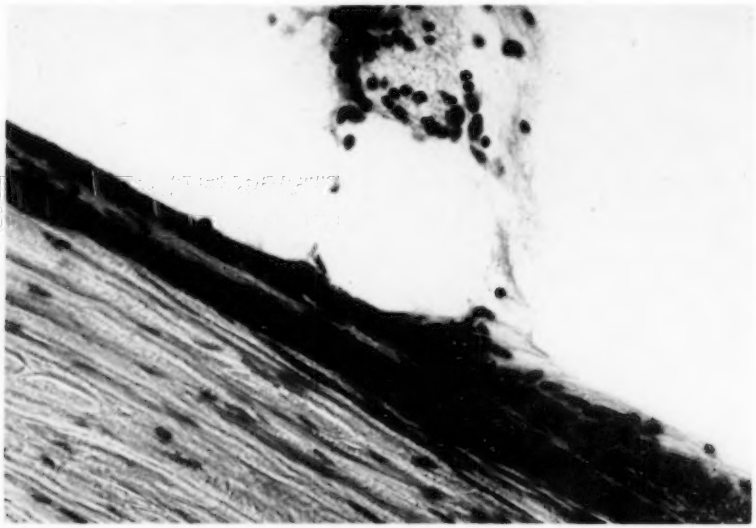

FIG. 32—Area "A" as indicated in figure 31. Small peripheral cysts that show the weakening of the inner and outer walls of this small cyst.

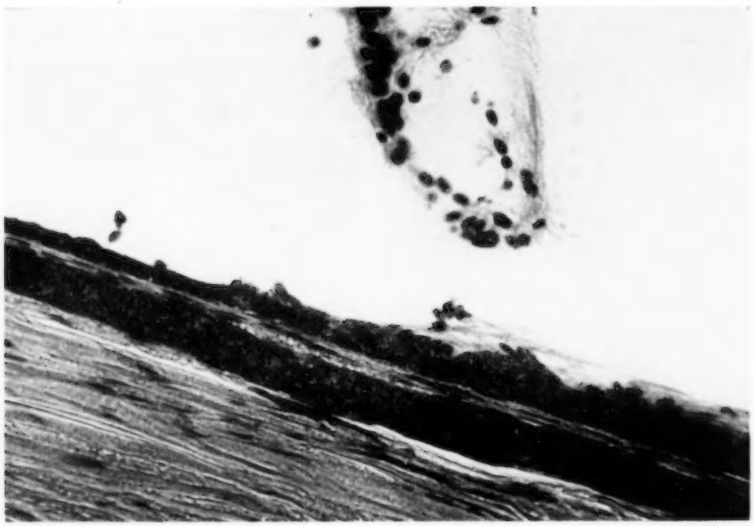

FIG. 33—The same area as in figure 31 but at a slightly different level which shows a through-and-through break in this cyst. The edges are sealed and there is no obvious traction, as can be seen by the sections studied.

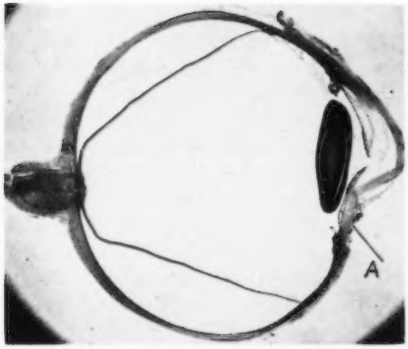

FIG. 34.—An eyeball which had a rupture of the globe near the limbus (A). In the area opposite to this there is an atrophic area in the retina which has resulted in a break.

subject because many times these cysts rupture and show no evidence of any vitreous traction and the base of the vitreous may be adherent in this area and seal the hole completely. This may prevent the fluid from migrating into the subretinal space.

A globe that contains small cysts in the periphery is shown in figure 31. The apparent detachment of the retina is an artefact as no subretinal fluid was seen beneath the retina grossly. There is no evidence of subretinal fluid microscopically. In a high power section of the area indicated in figure 31, we see a small peripheral cyst of the retina. Thin walls are present on the inner surface of the retina and there is a small break on the outer surface (fig. 32). The same area viewed at a slightly different level discloses a through-and-through break in the retina (fig. 33). With a detachment and shrinkage of the vitreous body it is conceivable that certain traction could be exerted on the lip of this break and cause the edges to invert. If this be the case, fluid could then migrate into the subretinal space and cause a detachment.

Certain perforating and nonperforating injuries exert a force to cause atro-

FIG. 35.—High power of area "A" in figure 34, showing the large retinal break.

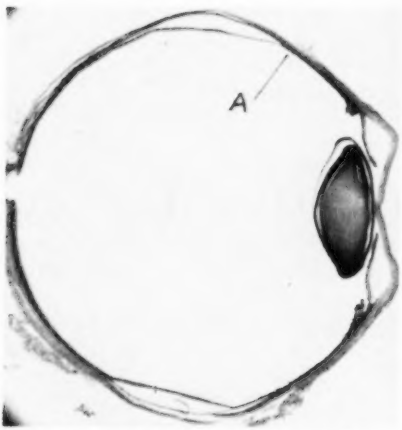

FIG. 36.—Injury of globe with atrophic area in retina "A"

phic changes in the retina that may finally result in a break. A blow on the eye may transmit the force to the opposite portion of the globe and later cause an atrophic area in the retina due to the contrecoup force. Figure 34 shows an eye with a history of a perforated injury near the limbus. Opposite this injury is a large break in the retina which is due to the degeneration at this point. A high power view of the point indicated in figure 34 shows the rounded sealed edges of the break (fig. 35).

On the other hand, if the force of an injury to the eyeball does not destroy the tissue but causes sufficient irritation that chorioretinal adhesions result, no detachment may form. Figure 36 shows an eyeball with an injury similar to that in the previous case, but sufficient chorioretinal reaction resulted so that the atrophic region of the retina was completely sealed by the chorioretinal adhesions. The high power view of area "A" shows the proliferation of the pigment epithelium as well as thickening of Bruch's membrane (fig. 37). The break is completely sealed and diathermy applications could hope for no better results.

Atrophic areas in the macular region often have the appearance of a break but with no detachment (fig. 38). Most of these are cysts or partial breaks. The inner layers may rupture but the outer layers may remain intact, and since there is no vitreous pull, the potentiality

FIG. 37.—Area "A" in figure 36 showing the chorioretinal adhesions following injury. These adhesions completely seal the break.

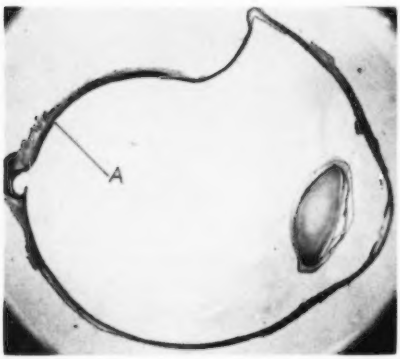

FIG. 38.—Partial break in macular region at "A."

of a retinal detachment is not great here. A high power view of the macular region of the globe shown in figure 38 demonstrates a partial break in the macula due to the rupture of the inner wall of a cyst that was present in the macular region (fig. 39). The outer layers of the retina can be clearly seen and therefore no detachment of the retina is present. Such a case as this would be very difficult to differentiate with the fundus slit lamp because of the overhanging lip of this atrophic area.

On the other hand, if there is a contracture of the inner surface of the retina by a proliferation of tissue on the inner surface of the retina, there may be sufficient traction to break the continuity. Figures 40 and 41 show a partial break in the macula due to the proliferation of a membrane on the inner surface of the retina. The contracture of this membrane has caused an eversion of the lips of this atrophic area and there is almost a through-and-through break. Further contracture of this membrane could very easily have caused a break with a subsequent detachment.

In many cases clinically it is possible to find numerous breaks in the retina where a detachment is present. The

FIG. 39.—Partial break in the macular region which appeared clinically to be a complete break.

FIG. 40—Partial break in macular region due to contracture of membrane on inner surface of retina.

horseshoe shape or linear break can be explained by traction but the multiple round holes must be caused in some other way. It has been shown that retinovitreous adhesions exist in the area of the break and there may well be other small atrophic points in the retina with adhesions to the choroid as well as to the vitreous. When the primary break is formed, fluid migrates beneath the retina. Sufficient force is then set up by the subretinal fluid, because of its momentum as well as its actual weight, to cause a greater area of the retina to peel off. If small chorioretinal adhesions exist, one of two things may happen: (1) the adhesions may be broken, or (2) the retina may give way. This would form a secondary break.

Figure 42 shows a detachment of long standing. Figure 43 is a high power view of the area indicated in figure 42. Here is shown force exerted on the retina from the subretinal space at a point where the retinal adhesion exists. Force at this point has caused the retinal elements to become stretched and distorted, and it is obvious that sudden motion of the eye could cause sufficient force to rupture this weakened spot and create a secondary break.

Figures 44 and 45 show a similar case. Here the retinal elements have been distorted by the traction at the point of the chorioretinal adhesion. The retina has been separated from this adhesion by an

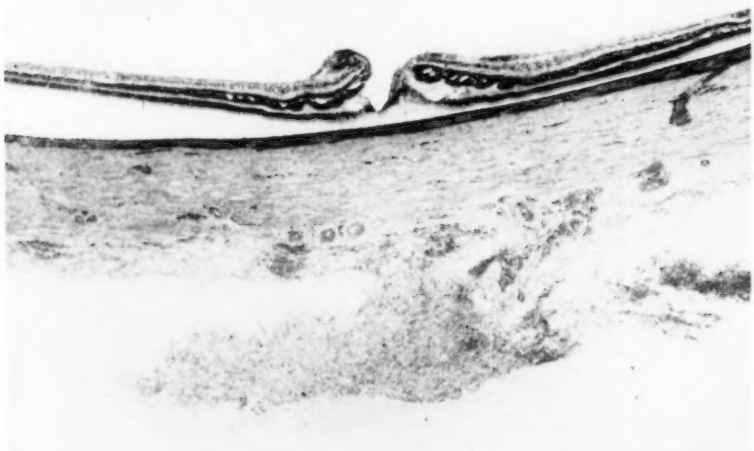

FIG. 41—Partial break in macula due to contracture of a membrane on the inner surface of the retina as shown in area "A" of figure 40.

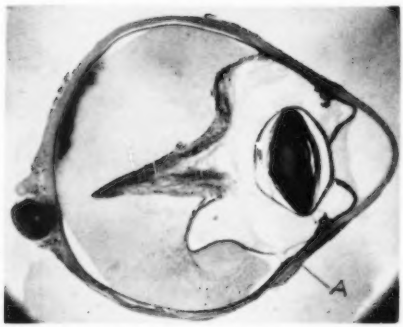

FIG. 42—Detachment of long standing with chorioretinal adhesions.

artefact but it is obvious that there has been traction at this localized area.

TREATMENT

Diathermy applications to the sclera cause shrinkage. Figure 46 shows the effect on the sclera three weeks after treatment. This flattening of the sclera can have considerable clinical significance. This particular case shows many of the points that have been previously discussed. Clinically, there was a bullous detachment of two weeks' duration. A horseshoe break was seen in the region of the equator. The diathermy applications were applied around the break with good flattening of the retina at the time of operation. Three weeks after the operation, ophthalmoscopic examination showed what appeared to be an elevated, bluish area which resembled a neoplasm. The eye was enucleated because of the possibility of a neoplasm.

Figure 47 (see Color Plate) shows the interior of the eye and demonstrates the break in the retina with the vitreous adherent to the lip of the break. The fine white areas on the retina are the result of the diathermy applications. Figure 48 is a microscopic section of the above case. This section shows clearly the flattening of the sclera in the area of treatment.

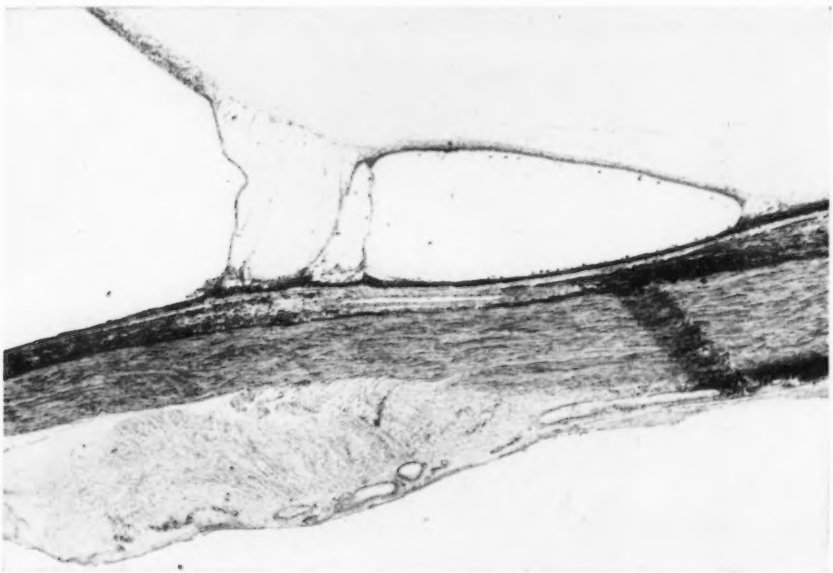

FIG. 43—Distortion of the retinal elements due to the force exerted on the retina by the subretinal fluid at a point of chorioretinal adhesion.

FIG. 44.—Detachment with chorioretinal adhesion.

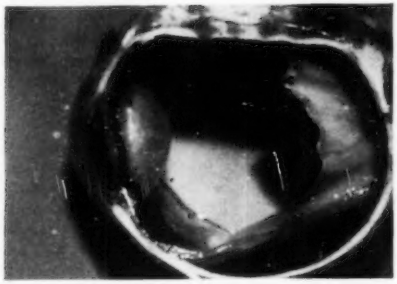

FIG. 46.—Flattening of sclera three weeks following diathermy.

Figure 49 shows a high power view at the area designated in figure 48. This section shows the region of the break. The lip of the break is still pulled in by the traction of the vitreous. There is some proliferation of the pigment epithelium. The retina appears to be well sealed surrounding the retinal break. In the choroid there is a large hemorrhage.

Figure 50 shows the area adjacent to the break and demonstrates the chorioretinal adhesions that sealed the break.

Figure 51 shows the periphery of the retina opposite to the area of the retinal break. Here one can see the peripheral cystic degeneration of the retina and there is also a dense vitreous strand which extends completely across the vitreous cavity. This vitreous strand can be traced to the lip of the break on the opposite side.

From the study of the microscopic sections in this case, it was apparent that the retinal break had been completely sealed. The ophthalmoscopic picture of

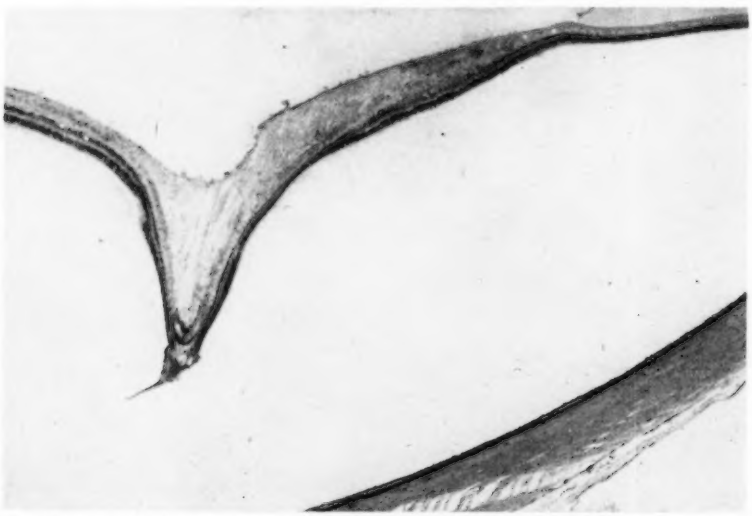

FIG. 45.—Distortion of the retinal elements due to force by the subretinal fluid at a chorioretinal adhesion.

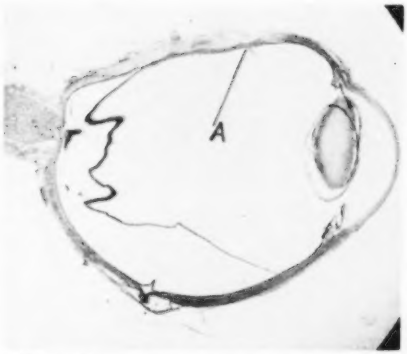

FIG. 48.—Microscopic section of detachment three weeks following diathermy. Marked flattening of sclera can be seen in region of treatment.

an elevated, bluish mass can be explained by the fact that the shrinkage of the sclera caused an apparent elevation and the bluish mass was due to the choroidal hemorrhage showing through the retina, which had become opaque due to the treatment.

It has been advanced by many observers that it is necessary to seal all of the breaks in the retina, otherwise the possibility of a complete reattachment is slight.

The next case was a bullous detachment of the retina below and a break was also found below. This area was treated by diathermy but the detachment persisted. Figure 52 shows a section of a globe with a detachment above. Figure 53 shows the area of treatment, designated as "A" in figure 52. This section shows the retinal break and the edge of the tear is pulled inward by the retracted vitreous. There is a proliferation of the pigment epithelium and some cellular elements are present in the choroid. It is obvious that this area has been well sealed. Figure 54 shows an area above opposite to the retinal break and designated as "B" in figure 52. Here one can see a retinal break. The lip of the break is attached to the vitreous and the edges are rounded and sealed, indicating its presence prior to enucleation. Since this break was not treated, a hope for complete reattachment could not be expected.

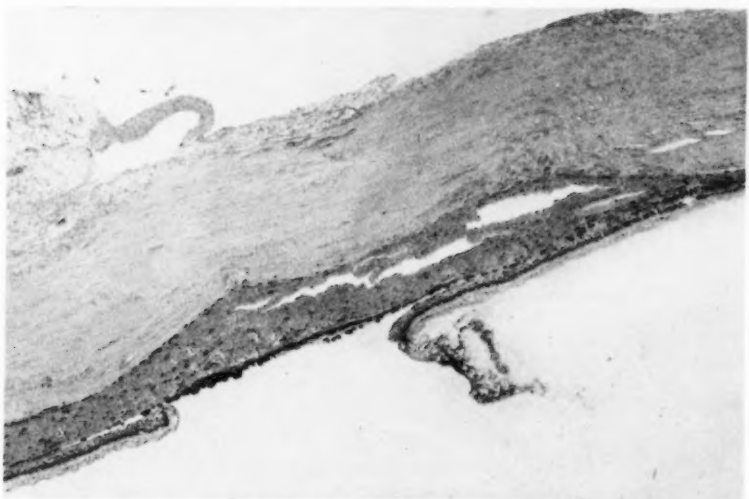

FIG. 49.—High power of a ca designated in figure 48 showing the edge of the retinal break attached to a vitreous strand. There is proliferation of the pigment epithelium with good chorioretinal adhesions surrounding the break. A large choroidal hemorrhage is present.

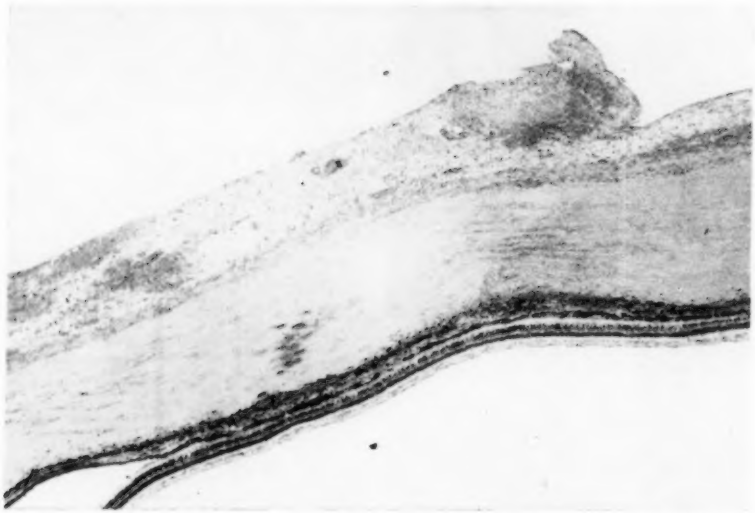

FIG. 50.—Area adjacent to the break demonstrating the chorioretinal adhesions following treatment.

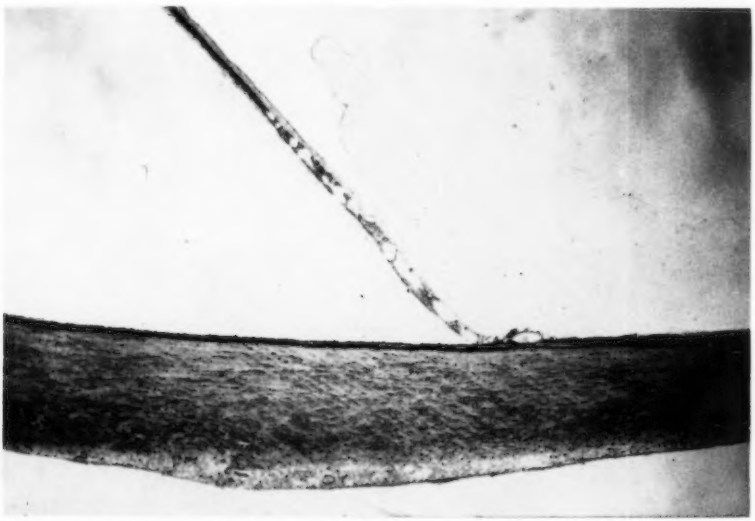

FIG. 51.—Periphery of retina opposite to retinal break demonstrating the contracted shrunken vitreous adherent to the periphery of the retina.

FIG. 47—Interior of eye showing retinal break and retracted vitreous adherent to lip of break. Vitreous on the left is adherent to lip of break at "A." The break at "B" is surrounded by faint white areas from the diathermy applications.

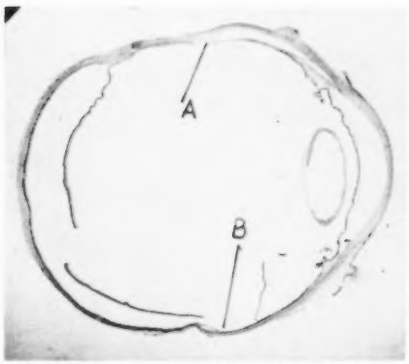

FIG. 52.—A section of a globe with a detachment of the retina two weeks following treatment. A break was found below and treated. A second break was also present above which was not treated.

A perforating diathermy may cause chorioretinal adhesions by two processes. First, the episcleral tissue may proliferate and grow down the perforation and cause a fibrous union of the choroid and retina. On the other hand, the pigment epithelium may proliferate to form the chorioretinal adhesions. From the study of my sections I favor the latter process. This can also explain the effect of the surface applications because here the episcleral tissue has no means of migration to form the union of the retina to the choroid.

Figure 55 is a perforating diathermy application which shows the downgrowth of the pigment epithelium forming a collar-button-like adhesion of the retina and choroid. The proliferated tissue in the episcleral region is made up of metaplastic pigment epithelium as well as fibrous tissue.

SUMMARY

Certain important pathologic processes pertaining to the detachment of the retina have been shown. The importance of the role played by the vitreous has been demonstrated by microscopic sections and an attempt has been made to show the sequence of events that takes place in the formation of a retinal detachment. The results and pathologic findings have been shown following diathermy application.

FIG. 53.—Area designated in figure 52. A retinal break below that has been treated. This shows the vitreous adherent to the edge of the break. There is a proliferation of the pigment epithelium following the treatment. Some edema and hemorrhage is present in the choroid adjacent to the retinal break. The sclera shows a loss of nuclear elements following diathermy.

FIG. 54.—Area "B" from figure 52 opposite to the retinal break that was treated. This section shows a retinal break with smooth edges and the vitreous is adherent to the lip of the retinal break.

FIG. 55.—A perforating diathermy application showing the proliferation of the pigment epithelium causing the chorioretinal adhesion.

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Symposium: Retinal Detachment

DIAGNOSTIC AND PROGNOSTIC FACTORS AS FOUND IN PREOPERATIVE EXAMINATION

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BY INVITATION

THE first symptom of a retinal detachment may be the sudden appearance of flashes of light. They indicate the existence of localized traction on the retina and precede or accompany either the formation of a retinal break or of a beginning detachment. When flashes of light result from this cause, they are often localized in one quadrant, generally below and nasally, and correspond to a retinal break in the opposite quadrant of the retina. However, contrary to expectation, the flashes may be in the same quadrant as the retinal break and incipient detachment. They appear suddenly, often disappear after a few days, and their cessation is frequently indicative of the extension of the detachment. More durable photopsias resembling lightning streaks, which appear over the temporal field and are vertical or circular in direction, are often of benign prognosis.[1] Light flashes occurring in early cases of retinal detachment are often accompanied by a sudden increase in vitreous floaters or by a shower of black dots. Another early symptom is the sudden appearance, in the patient's field of vision, of brownish spots and threads caused by a vitreous hemorrhage. This hemorrhage usually results from the rupture of a retinal vessel by a recent retinal break. Because it is either small or moderate in amount, it seldom causes a completely black reflex. Obscuration of fundus details may nevertheless be such that it is necessary to wait for the hemorrhage to be absorbed before a diagnosis of retinal detachment can be made and the retinal breaks can be found.

In all cases of vitreous hemorrhage, particularly when the bleeding is not massive, it is extremely important to consider retinal detachment as a definite possibility until there is some evidence to the contrary. A detailed history will reveal whether the patient had flashes of light around the time the hemorrhage occurred and whether he ever had a retinal detachment in either eye, as such a history would be suggestive of an early detachment. Both fundi should be carefully examined in the hope of detecting the possible cause of the vitreous hemorrhage. Evidence of diabetic retinopathy, of Eales' disease or of active choroiditis does not rule out the existence of a primary retinal detachment since detachment has repeatedly been observed in eyes affected by one of these conditions. When a vitreous hemorrhage is present, it frequently happens that the diagnosis of retinal detachment is not made until a large detachment has formed. There is no doubt that a more careful fundus examination of such cases would often permit a much earlier diagnosis (fig. 1).

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The symptoms described so far generally accompany the formation of retinal breaks, which are the first step toward a retinal detachment. This may occur almost immediately or it may be delayed for months or even years after the breaks have formed. Rare cases have been observed where the retina never became detached after the formation of retinal breaks. When a fairly large detachment is present, it causes a gray cloud or veil in the visual field. It is important to determine in what part of the visual field the cloud first appeared, for retinal tears will often be found in the sector of the fundus corresponding to the earliest field defect. The appearance of a veil or cloud in the visual field often coincides with the disappearance of light flashes. In quite a number of cases, the early stages of retinal detachment are asymptomatic and field loss is the patient's first complaint.

It is well known that between 40 and 60 per cent of all eyes with detachment are myopic. Approximately 20 per cent of the detachments seen at the Retina Service of the Massachusetts Eye and Ear Infirmary occur in aphakic eyes. Although myopia and aphakia predispose to retinal detachment, these conditions do not, by themselves, affect the prognosis. It is the degree of peripheral degeneration found in these cases which is important.

In general, the intraocular tension is diminished when a retinal detachment is present, with very low tensions usually accompanying large detachments. A moderate hypotension is not an important prognostic sign. Although intraocular hypertension with a retinal detachment may suggest a choroidal tumor, it should be remembered that primary retinal detachment may occur in glaucomatous eyes. Moreover, deep chamber glaucoma may be a complication of retinal detachment, particularly in retinal dialyses (disinsertions) of long standing and in detachments due to alterations of the ora serrata region. The association of glaucoma with primary retinal detachment has no particular prognostic significance as far as the retinopexy is concerned. The diathermy applications that are made in order to close the retinal breaks frequently bring about a decrease of intraocular tension for a period of several months. Sometimes this decrease is permanent.

Slit lamp examination may supply useful information. Signs of moderate anterior uveitis are fairly frequent and do not affect the prognosis. Active uveitis may coexist with a primary retinal detachment. In such cases the prognosis is comparatively favorable and operation is not contraindicated unless the inflammation is intense. If with retro-illumination the posterior pigment layer of the iris appears atrophic, particularly near the pupillary border, considerable degenerative changes in the fundus periphery may be expected. In these cases, pigment deposits may be observed on the back of the cornea, in the aqueous, on the lens capsule and in the anterior portion of the vitreous. Posterior cortical and posterior subcapsular lens opacities are frequently associated with alterations in the region of the ora serrata.

A great deal of work has been done on the clinical appearances of the vitreous body in cases of retinal detachment. Observation of the deep portions of the vitreous body with a slit lamp requires the use of a flat contact lens or of a minus 55 diopters concave lens in front of the eye, and also a device to reduce the angle between the observation axis of the microscope and the illumination axis of the slit lamp. In most cases of idiopathic detachment, the vitreous is degenerated, and frequently an alteration of the vitreous, known as vitreous detachment, is observed (fig. 3). This means that a portion of the vitreous cav-

ity, generally the posterior and upper portion, appears optically empty, whereas the anterior and lower portion contains loose threads and membranes. The limit generally appears as a wavy grayish line which may only be detected by careful observation. It is likely that this condition is the result of a collapse of the vitreous gel. However, it is the writer's opinion that most cases result from the formation of an optically empty cavity inside the vitreous body and not between it and the retina. In most cases, therefore, the hyaloid membrane and a cortical layer of vitreous probably remain adherent to the retina. The presence or absence of a vitreous detachment seems to affect the prognosis very little. It should be noted that vitreous detachment frequently occurs in cases where no retinal detachment is seen and its value as a precursor sign of retinal detachment has not been definitely established. In rare cases, slit lamp examination may disclose a translucent membrane stretched across the whole fundus of the eye, located about the middle of the vitreous cavity and roughly parallel to the posterior lens capsule (fig. 3). This membrane is smooth and does not move with movements of the eye. It appears considerably more opaque than the wavy membrane described above and is as easily discernible as the posterior surface of the lens. The space behind it appears optically empty. Its attachment, along the equator of the retina, produces a fixed circular fold in the retina. It is the writer's opinion that this membrane is the posterior hyaloid. The prognosis of cases showing such a hyaloid membrane is serious.

In practically all cases of retinal detachment, visual fields show impairment. Absolute field defects generally correspond to areas where the retina is very elevated or where the detachment is of long standing. Relative defects are often caused by flat detachments. It should be emphasized that a flat detachment may not cause any indentation of isopters mapped out with large or brilliant test objects; therefore, it is important to use small test objects or reduce the illumination of the perimeter. A temporal detachment which is confined to the extreme periphery and does not extend further towards the posterior pole than the equator does not cause field defects with any test object. The same is true of some cases of early detachment where retinal breaks are associated with a minimal amount of detachment. Consequently, the absence of field defect does not rule out the existence of a very peripheral detachment or of retinal breaks surrounded by a minimal amount of detachment.

Examination of the fundus with the ophthalmoscope is of greater importance than perimetry because it permits detection of retinal breaks; it also gives other important information which affects the prognosis. Whatever method of ophthalmoscopy is used, it is important to have an instrument which is versatile and which provides the strongest illumination available. When a very highly myopic fundus is to be examined, one should use an instrument containing a sufficient range of minus lenses. A direct ophthalmoscope with powerful illumination is particularly useful for the examination of small lesions located between the posterior pole and the equator. When examining an emmetropic eye with a direct ophthalmoscope and without indenting the sclera, the fundus can be seen clearly up to a region which, in most cases, is only slightly anterior to the equator. This means that approximately one third of the total surface of the retina cannot be seen clearly with this method. Statistics of the Retina Service at the Massachusetts Eye and Ear Infirmary, show that a great number of retinal breaks, located in the extreme fundus

periphery, cannot be detected with a direct ophthalmoscope without scleral indentation. For this reason the writer has developed an indirect ophthalmoscope which, in many cases, enables the observer to see the retina from the disc up to the posterior border of the ciliary processes. The instrument used was described before this Society in 1950.[2] It has a powerful system of illumination, it permits stereoscopic visualization of the fundus, and it is so built that a maximum advantage can be derived from the use of the Trantas[3] method of examining the extreme periphery. This method consists of indenting the sclera in the ciliary region or slightly posterior to it in order to bring into view structures which are otherwise invisible. Direct ophthalmoscopy is impractical when the sclera has to be indented, because the observer is too close to the eye under examination and finds it difficult to control the movements of the instrument used for indentation. With the new indirect ophthalmoscope,* the observer is 30 centimeters away from the eye under examination—a good working distance. He can move freely in all directions, since the ophthalmoscope is worn on his head. His right hand is free at all times and can be used to indent the sclera with a small instrument, mounted on a thimble, and called scleral depressor. The technic of scleral indentation was also described before this society in 1950.[2]

Fundus examination of a case of retinal detachment should be performed in a darkroom under maximum mydriasis, obtained either with drops or with a subconjunctival injection. The writer prefers to use phenylephrine powder in the conjunctival sac, followed by instillation of 1 per cent homatropine, 0.5 per cent atropine or 0.2 per cent scopolamine. For subconjunctival injection, 0.2 cc. of the following mixture is used:

Phenylephrine hydrochloride	25 mg.
Homatropine bromhydrate	20 mg.
Procaine hydrochloride	50 mg.
Aq. dist.	5 cc.

Ophthalmoscopic examination should supply the answers to the following questions:

1. Is the retina detached?
2. What is the extent of the detachment?
3. Where are the retinal breaks?
4. What is the status of the macula?
5. What is the status of the vitreoretinal adhesions?

1. *Is the retina detached?*

By illuminating the fundus with any type of ophthalmoscope at a distance of two feet or so, the quality of the red pupillary reflex can be judged (fig. 4). If this is poor everywhere or has turned gray in one sector, there is a possibility of retinal detachment, and a careful ophthalmoscopic examination of the fundus should be performed. However, it should be noted that a flat retinal detachment may not affect the red reflex. A detached retina generally appears like a translucent gray veil making the choroidal pattern appear fuzzy, whereas a retina in situ is transparent except for the retinal vessels. The gray veil is identified with the retina because it contains the retinal vessels. It generally forms numerous folds which move freely with movements of the globe. The vessels, therefore, appear more tortuous and of a darker color than normally.

In case of smooth and flat retinal detachment, diagnosis may be missed unless attention is paid to the following details (fig. 20): The retinal vessels are not tortuous or overly dark, but if a strong light is used, they will cast a shadow on the underlying choroid. The slight haze formed over the choroidal pattern by the flatly detached retina may appear smooth under direct illumi-

* Manufactured by the American Optical Company.

nation, but often has a cobblestone appearance if illuminated by light reflected from the choroid. This indirect illumination may be obtained by casting the beam of the ophthalmoscope light slightly to the side of the region to be examined.

Diagnosis of retinal detachment in difficult cases is further discussed under paragraph 3.

2. What is the extent of the detachment?

The extent of the detachment is not in itself of much prognostic significance, but it often gives an indication as to where the retinal breaks are located (fig. 21). The following general rules may be useful as a rough guide in cases where the detachment is not of very long standing. If one upper quadrant only is affected, breaks are probably present in the superior part of the detachment (fig. 21A). If both upper quadrants are equally affected, breaks are often found near 12 o'clock (fig. 21B). If the detachment is below the horizontal meridian and one or both of the inferior quadrants are affected, breaks will probably be found either along the meridian bisecting the retinal detachment (fig. 21, C and D), or in the upper portion (fig. 21, E and F). If an inferior detachment extends considerably above the horizontal meridian, there is practically always a break in the upper part of the detachment. When a break in an upper quadrant has been present for over four weeks, the subretinal fluid often sinks in the lower half of the fundus, where large balloons of detachment may form, whereas in the upper quadrant, which contains the original retinal break, the detachment becomes very flat and may easily escape notice (fig. 21G). If all quadrants are affected, it generally means that breaks are present in different sectors of the fundus or else that breaks are present near 12 o'clock (fig. 21H). It is generally considered that a total detachment has a poor prognosis. This assumption is only partly correct, since a total detachment is as liable to become reattached as a partial one, provided that all the retinal breaks are detected and repaired. However, detachments of long standing and recent detachments with large or numerous retinal breaks tend to become total and have a poor prognosis. These two facts may be responsible for the poor prognosis generally made in all cases of total detachment.

It should be remembered that in any of the above-described detachments, breaks may be observed in several meridians. Breaks may even be present in areas where the retina is in situ (fig. 2).

The limits of the detachment may give an indication as to its duration. If the limits between the detached and nondetached retina are marked by a whitish gray or by a pigmented line, called demarcation line or friction line (figs. 4 and 22), this indicates that the detachment has been present and fairly stationary for at least three months and probably for much longer. A demarcation line generally forms an arc with the largest retinal break near its center. Such a line is often associated with breaks in the inferior half of the fundus, particularly cases of dialysis in the young. Several more or less concentric demarcation lines may be observed; when they are incomplete, they are more marked at the extreme periphery. As the presence of a demarcation line indicates that the detachment is not recent, the degree of functional improvement after operation is often disappointing.

3. Where are the retinal breaks?

If the detached retina has a break in its continuity, the choroid is more clearly visible through it. Generally, therefore, a retinal break appears as a sharply outlined red spot in which the choroidal pattern is easily seen. There is a

marked contrast between it and the surrounding grayish detached retina. Care should be taken not to confuse a retinal break with a retinal hemorrhage. In most cases this confusion is easily avoided by paying attention to the following details: If the red spot is a hemorrhage, it is generally of a darker and more homogeneous color than the choroid. The granular red color of the choroidal pattern showing through a retinal break is in the plane of the choroid, whereas the smooth, even, dark coloration of a retinal hemorrhage is best seen when the ophthalmoscope is focused on the plane of the detached retina. This difference in level is readily visible with a stereoscopic ophthalmoscope or by the observation of parallactic displacements. In the case of a retinal break there is a parallactic displacement of the red area in relation to retinal details, while in the case of retinal hemorrhage there is no such displacement.

The characteristics described so far help in making a diagnosis of retinal detachment and of retinal break in simple cases. There are many instances, however, where an accurate diagnosis cannot be arrived at without more detailed investigation. The gray veil formed by the detached retina is sometimes inconspicuous, particularly if the red background of the choroid is discolored by some degenerative process that also causes the retina to be extremely thin. The same difficulty occurs if the transparent media form a gray haze, as in cases where the vitreous, lens or cornea contain diffuse opacities. Such opacities of the media often make the retina appear as if there were a flat detachment when in fact there is none, since they cause the choroidal pattern to appear fuzzy and the retinal vessels to look dark. Differential diagnosis between real detachment and the appearance caused by hazy media may be helped by the use of stereopsis and by observation of parallactic displacement of the retinal vessels against the choroidal background. If monocular ophthalmoscopy is used, observation of parallactic displacement alone is relied upon.

When diffuse opacities of the media are present in a case of retinal detachment, the contrast between retinal break and detached retina is greatly reduced. Similarly, when a detachment is so flat that there is very little color contrast between the detached and attached part of the retina, retinal breaks often cannot be found. The most important adjuncts for the diagnosis of retinal detachment as well as retinal breaks, when there is lack of contrast, are the use of a strong light, for direct and indirect illumination, and the use of a scleral depressor. Scleral indentation of an area where no detachment is present produces an artificial elevation in the fundus which is bordered, toward the disc, by a shadow forming a narrow, dark band. If a flat detachment is present, indentation permits the observer to visualize it at a very oblique angle, making it less transparent and increasing the color contrast between the retinal breaks and their surroundings (fig. 10). Scleral indentation also causes small folds to appear in a smooth and flat detachment, particularly in the area of the narrow, dark band. If indirect stereoscopic ophthalmoscopy with scleral indentation is used, it has been the writer's experience that one or several breaks are found in 99 per cent of all cases of idiopathic detachment.

The great majority of retinal breaks are found either near the equator or near the ora serrata, although in a few cases breaks are posterior to the equator. Equatorial breaks, which are often located in the upper temporal quadrant, may be of two morphologic types. The first type includes horseshoe-shaped tears with a flap and oval tears with an operculum floating in the vitreous (fig. 8). The second type includes round, oval

or irregular holes which are nearly always several in number, and are found in markedly atrophic areas. Retinal holes are often located inside areas of lattice-like degeneration, whereas horseshoe-shaped tears may be found on the edge of such areas (fig. 16). Lattice-like degeneration is characterized by white lines arranged as a lattice, many of which are either retinal vessels surrounded by a white sheath or completely obliterated vessels. Equatorial breaks occur more frequently in myopic eyes than in nonmyopic eyes and often result from chorioretinal degeneration near the equator. When equatorial degeneration is advanced, as in many high myopes, it tends to bind the retina down to the choroid. In such cases, a detachment may form between the equator and the ora serrata and be prevented from extending toward the posterior pole by chorioretinal adhesions. Such a detachment may remain undiagnosed for many years, and when it finally extends toward the posterior pole, it may be a detachment of very long standing (fig. 19).

A large number of cases show breaks in the region of the ora serrata. For adequate examination of this region, strong illumination, stereopsis, and scleral indentation are almost always required. The frequent use of scleral indentation will make the observer familiar with the normal appearances of the ora serrata. It forms a pigmented line, normally more scalloped and much narrower nasally than temporally (fig. 14). Each scallop is called an ora bay and the pigmented projections which extend anteriorly and separate the bays from each other are called the ora teeth. It should be remembered that near the ora serrata, the retinal vessels normally appear darker and more tortuous than around the posterior pole, and that parallactic displacements are not very helpful because of interference from irregular astigmatism.

If a detachment is present near the ora serrata, it is generally diagnosed without difficulty when the sclera is properly indented (fig. 18). By comparison with another portion of the extreme periphery of the same eye, a detached area near the ora serrata looks whiter, and folds appear in the detached retina as a result of indentation. The shadow bordering the indented area toward the disc does not form a sharply outlined dark band but becomes grayish and rather indefinite. When the retina is detached at the ora serrata itself, examination of this region in profile shows that the apparent thickness of the retina is much increased.

All the breaks occurring in the ora serrata region have one common characteristic: the detachment they cause often progresses slowly and tends to affect the posterior pole only at a late stage. In a few cases, these breaks are of the same type as those found near the equator, but most often they are of one of the following types: small and multiple, generally in patients who are over 45 years of age; retinal dialyses in young patients; or traumatic retinal dialyses. The majority of the breaks observed in the first type either form small retinal dialyses affecting only one ora bay or are related to meridional folds. A meridional fold is generally observed over an ora tooth (fig. 7) but occasionally occurs on the convex border of an ora bay (fig. 17). Breaks related to meridional folds may appear round or crescentic, either in the fold itself or at its posterior extremity. These lesions are often found in the upper half of the fundus, with the upper nasal quadrant perhaps most frequently affected. They are generally associated with marked chorioretinal degeneration near the ora serrata, or with quiescent and sometimes

with active peripheral uveitis,* posterior cortical cataract, and occasionally with deep chamber glaucoma. It is the type of fundus pathology most frequently seen in aphakic eyes with detachment. The prognosis in such cases is good, provided all the breaks have been discovered and provided there is not too much chorioretinal degeneration.

The second type of break in the region of the ora serrata is the single retinal dialysis occurring in young people, which predominantly affects the lower temporal quadrant and has a tendency to be bilateral (fig. 6). Such a dialysis occupies several ora bays and, quite frequently, a whole quadrant. The disease may be stationary over long periods, causing the formation of demarcation lines. The prognosis is generally good, although, because of the long standing of the detachment before operation, the degree of visual recovery is sometimes disappointing. In rare cases the dialysis extends all around the globe, making the prognosis hopeless.

Disinsertions or retinal dialyses resulting from a direct nonperforating injury of the globe (fig. 9) are generally located either above and nasally or below and temporally. In a number of cases, the narrow insertion of the base of the vitreous to the ora serrata and pars plana is torn as a result of the injury and floats freely in the vitreous cavity. Avulsion of the base of the vitreous is probably the direct cause of a certain number of traumatic retinal dialyses. The edge of many traumatic retinal dialyses remains close to the choroid so that it is impossible to see them unless the sclera is markedly indented; once they have been found, however, surgical treatment is generally successful.

*Peripheral uveitis is characterized by foci of inflammation in the region limited posteriorly by the equator and anteriorly by the posterior border of the ciliary processes. It often follows a protracted course and is generally asymptomatic.

The search for retinal breaks should be systematic. A good method is to have the patient seated and to follow every retinal vessel from the disc to the extreme periphery. A sketch should be made of any breaks or suspected breaks. The writer uses a special chart for this purpose, which has been described before this society in 1950.[2] When examination in a sitting position is completed, the patient should lie down on a wheel stretcher and be re-examined. Any doubtful area near the equator should be studied carefully, using scleral indentation, and the region of the ora serrata should be examined with a scleral depressor. At the first examination, retinal breaks may not be found, as they may be hidden by retinal folds. In such case, the patient should be re-examined in both a sitting and lying position and always in a darkroom under maximum mydriasis. Examination should be repeated daily for several days, and areas showing visible chorioretinal degeneration should be scrutinized with particular care. There is a natural tendency for the observer to be satisfied as soon as one retinal break has been found, in spite of the fact that cases with only one break are in the minority. In general, several breaks are found in the same area although it is not exceptional to find them in different quadrants. The writer has found as many as twenty-nine breaks in one eye. It can be quite certain that all visible breaks have been detected only when every part of the retina, whether detached or not, has been adequately examined. A particularly careful search for additional breaks should be made in cases where those found do not explain the extent and location of the detachment.

When the macula is not yet detached, it is important to determine how much the detachment threatens to involve the macular area. The speed at which a detachment extends towards the posterior

pole depends mainly upon the size and number of the breaks and upon their distance from the posterior pole. In case of equatorial breaks, for instance, the detachment generally involves the posterior pole more rapidly than when the breaks are near the ora serrata. The quadrant in which the breaks are located is also important: if they are in the upper half of the fundus, the detachment invades the macular area more rapidly than if they are in the lower half, and if they are temporal, the macular area will be involved sooner than if they are nasal.

When the macula is detached, the prognosis is made poorer still if the breaks are large or numerous, particularly if some are located in the vicinity of the posterior pole.

4. *What is the status of the macula?*

If the macula is detached, it nearly always appears edematous, while its yellow color is more marked and affects a wider area than in a normal macula. The visual acuity drops considerably, with a flat detachment of the macula affecting the visual acuity less than one that is very elevated. If the macula has been detached even slightly, recovery of all visual acuity that was lost is unlikely.

In some cases, a macular cyst is present which may resemble a round macular break, making differential diagnosis between the two extremely difficult (fig. 11). A macular cyst generally has a thin internal wall that is visible when using slit-like illumination as provided by the Friedenwald ophthalmoscope, the slit lamp and a flat contact lens or one of the large binocular ophthalmoscopes. When this internal wall is completely visible, it is certain that no macular break is present. However, if the internal wall is not visible, it should be remembered that it may be ruptured, leaving the external wall of the macular cyst intact. Such a defect forms an incomplete macular break. The fact that the choroidal pattern is seen clearly through the macular defect, and that the choroid is obviously at a different level than the edges of the defect, is no definite proof of complete macular break. Differential diagnosis between incomplete and complete macular break is always difficult and sometimes impossible to make. A complete break has a "punched-out" appearance with sharp limits. A macular cyst often has ill-defined limits and is frequently surrounded by other smaller cysts (fig. 11C); these are made more readily visible by indirect illumination. In some cases, a flap or an operculum is visible and makes the diagnosis of macular tear certain. Macular cysts are often observed in the absence of a macular detachment whereas a complete macular break is accompanied with at least a very flat macular detachment (fig. 11B). The extent of the detachment may also give important indications as to the nature of the macular defect. If the detachment does not affect the periphery but is limited to the posterior pole, the suspected macular break may well be the only break present. If the detachment extends to the periphery, the presence of peripheral breaks should be suspected and this makes a diagnosis of macular break more doubtful. It is important to remember that macular breaks seldom occur, and that there are probably many macular lesions being treated as breaks when such is not the case. The writer has not found them in more than 1 per cent of 400 consecutive retinal detachments. It is obvious that as diathermic treatment of a real macular break will give the patient poor central vision, even in successful cases, such treatment should be undertaken only if the diagnosis is well established.

Certain cases of retinal detachment, showing a marked degree of peripheral degeneration, have a tendency to macular degeneration as well. Even when successfully operated on, such cases

sometimes develop macular degeneration months or years after the operation. In making a visual prognosis, this possibility should always be taken into account.

5. *What is the status of the vitreoretinal adhesions?*

Apart from the detection of retinal breaks, which is of paramount importance for the treatment of retinal detachment, it is also necessary to note any other details in the fundus which may help in making a more accurate prognosis. Any signs of the extent and course of the vitreoretinal adhesions and of the amount of vitreous shrinkage are of great importance. In this connection, the following fundus appearances are significant: fixed folds, massive vitreous retraction, and breaks with a rolled-up edge.

Ordinarily, retinal folds move freely when the eye moves and they change markedly in shape and location when a seated patient lies down or vice versa. When retinal folds are free, the detachment often subsides considerably with strict bed rest, provided the eye is immobilized in the proper position with the breaks dependent. Retinal folds which do not move when the eye moves are called fixed folds. Any movement seen in such folds is not more than a slight tremor, as if they were imprisoned in a jelly. Fixed folds do not flatten out with bed rest, and it is generally assumed that the vitreous is adherent to them. There are two types of fixed folds which are of particular interest. The first type is a circular fold, which is in the region of the equator and roughly parallel to it, forming a sharp ridge in the fundus; it may extend around the whole circumference of the globe. In some cases, it is possible to see the stretched hyaloid membrane attached to it (fig. 3B), and in other cases, visible vitreous strands seem to be pulling on it. It may also be associated with massive vitreous retraction (fig. 15) or with tears having a rolled-up convex edge (fig. 12). The second type of fixed fold is the star-shaped fold (fig. 13) which may be found in any part of the globe, generally, however, below the horizontal or in the macular area. Several of these may be present in the same eye.

Fixed folds make the prognosis poor. If they are small or affect an area which is remote from the retinal breaks, a permanent cure may be effected. The same is true if they are associated with very small breaks. When the fixed folds or the retinal breaks are large or when they are located near each other, failure of retinopexy generally follows. Sometimes the whole retina seems to form one large star-shaped fold around the disc; this is called massive vitreous retraction (fig. 15). As far as it may be determined, the hyaloid in such cases adheres to the retina and shrinks, causing a detachment that rapidly becomes total. There are two principal ophthalmoscopic characteristics of massive vitreous retraction. The first is that all the retinal folds are fixed; and the second is that the fixed folds irradiate from the posterior pole of the globe towards the equator. In many instances, a circular fixed fold is present as well. Folds irradiating from the disc have rounded apices near it that are gradually pulled toward the anteroposterior axis of the globe. This displacement soon causes the round apices to have an overhanging edge that may hide the disc completely. Massive vitreous retraction is incurable, although after it has been present for several months it often loses its characteristic appearance. These cases never improve by bed rest. If the subretinal fluid is allowed to escape and saline solution is injected into either the anterior chamber or the vitreous, the retina flattens out somewhat but the folds retain their general shape. In the writer's experience, all cases of true massive vitre-

ous retraction have so far resisted therapeutic attempts, including scleral resections.

Fixed folds and massive vitreous retraction are found more frequently in detachments of long standing; this is probably the main reason why old detachments can seldom be cured. However, recent cases are sometimes affected, and seemingly there is no way to predict when an eye will be affected and when it will not. Massive vitreous retraction has been seen to occur postoperatively, which probably means that the surgical procedure itself is a precipitating factor. Vitreous hemorrhage also seems to be a precipitating factor, particularly when fairly large. It is impossible to decide whether it is the bleeding itself which is responsible, or whether bleeding merely indicates that vitreous shrinkage follows a particularly malignant course.

In cases of massive vitreous retraction, the tension is often soft and the vitreous hazy; the pupil dilates poorly, the aqueous shows flare and contains many clumps of pigment. Attacks of ciliary pain, posterior synechias, cataracta complicata, and secondary glaucoma are not infrequent.

Another characteristic appearance which probably indicates extensive vitreoretinal adhesions, making the prognosis guarded, is the presence of retinal tears with rolled-up convex edges (fig. 12). They are generally located at the equator or slightly anterior to it, where several tears of this type may form a row. Tears with rolled-up convex edges have straight anterior borders and no flap is attached to them. A fixed circular retinal fold is often found on the anterior border of the row of tears. It is not exceptional to see such tears increase rapidly in size and number even though the patient is on strict bed rest, and massive vitreous retraction has developed in several instances.

Once the retinal breaks have been located and the fundus thoroughly investigated, it is important to try and induce absorption of the subretinal fluid by bed rest. The patient should have both eyes bandaged in order to avoid ocular movements. His head should be kept in such a position that the retinal breaks are in the dependent part of the globe; however, this position should be relatively comfortable. Experience has shown that there is no advantage in having the foot of the bed elevated. Bed rest with the retinal breaks in a dependent position tends to relax the pull exerted by vitreous bands or by the collapsed vitreous on the retina. The choroid absorbs the subretinal fluid, and relaxation of traction on the retina allows it to become reapplied against the choroid. As a rule, this result cannot be achieved unless strict bed rest is enforced, keeping the head and eyes as immobile as possible. The patient is considered to be ready for operation when no further flattening out of the detachment may be expected. All the improvement that may be hoped for from bed rest is generally obtained after one to seven days. More than seven days of bed rest are useful only in exceptional cases; usually three or four days will suffice. In elderly or restless patients, it is advisable to shorten the preoperative rest period. If the retina becomes completely reattached after bed rest alone, such apparent cure does not, as a rule, last more than a few days. Permanent cures after bed rest have been recorded, but are exceedingly rare. A marked improvement is of good prognosis, but it should be remembered that massive vitreous retraction may occur postoperatively, in cases improved by bed rest, and ruin the operative result. The absence of improvement makes the prognosis poor but not hopeless. In most cases with either fixed folds of any kind, or massive vitreous retraction, or

tears with rolled-up convex edges, improvement from bed rest is slight or absent. Such failure to improve may also be caused by the inability of the choroid to absorb the subretinal fluid or by the large size and number of the retinal breaks. In any case, where the detachment or the size or number of the breaks increase in spite of strict bed rest, the prognosis is very poor.

One cannot overemphasize the importance of early diagnosis of retinal detachment. Operation before the macula is involved is essential for the preservation of good visual acuity. When the macula is detached, immediate operation is important to salvage as much of the central vision as possible and to avoid fixed folds and massive vitreous retraction. In some cases, it is possible to detect retinal breaks even before the detachment has caused any functional disturbance. Such findings would be more frequent if the fellow eye were carefully examined in every case of retinal detachment and if close attention were paid to the early signs and symptoms of retinal detachment. The surgical prognosis in very early cases is nearly always excellent.

Differential diagnosis between primary retinal detachment, retinocele* and malignant choroidal tumor will be briefly discussed.

Clear fluid may accumulate in the inner nuclear or in the outer molecular layer of the retina, forming an intraretinal collection of fluid and creating the appearance of a smooth and bullous detachment, more marked or entirely located in the periphery of the inferior temporal quadrant. In exceptional cases, such a retinocele may reach the posterior pole. It causes a field defect which is clear cut and has steep limits. The internal layers of the retina, which contain the large retinal vessels and form the extremely thin and smooth internal wall of the retinocele, are pushed inward. Sometimes this internal wall is so thin and transparent that only the retinal vessels are visible. In many cases, the vessels seen in the affected area are either narrow or completely obliterated, while the internal surface of the retinocele shows glittering spots that look like snowflakes. These are important diagnostic features. Larger shiny areas, which are light reflections on the internal surface, are frequently the first sign which attracts the observer's attention and their diagnostic importance lies in the fact that they may be spotted even through an undilated pupil. Round or oval breaks may be observed in the internal wall and do not seem to cause a real retinal detachment so long as the external wall contains no break (figs. 2 and 4).

A retinocele shows no retinal folds and does not change in appearance when the patient changes his position. It may be egg-shaped and sharply outlined, in which case it is called a retinal cyst (figs. 2 and 4). It is probable that these retinal cysts have a glial lining. In other cases, a retinocele contains one or two peripheral egg-shaped balloons which are connected by a flat elevation extending all around the extreme periphery (fig. 17). This is called a retinoschisis,* or splitting of the retinal layers. In the case of a retinoschisis, no glial lining is expected, and the occurrence of a real detachment is feared much more than in the case of a retinal cyst.

As long as retinoceles show signs of very slow progress, as demonstrated by visual fields taken every three to six months, no treatment is indicated. However, a real detachment may develop which will be indicated by the following signs: moving retinal folds appear in the fundus, the field defect becomes

*From the Greek word *kele*, which means tumor, hump. The word retinocele is analogous to mucocele, meningocele, hydrocele, etc.

*From the Greek word *schisis* which means split, separation.

more extensive and acquires sloping limits, instead of the usual steep ones; retinal breaks are visible either in the area affected by the retinocele or elsewhere. As mentioned earlier, a break of the internal wall seems to cause no detachment so long as the external wall is intact, but if a break forms in the external wall, detachment follows. Such a break may be difficult to detect; it is almost always located near the ora serrata where it forms an incomplete retinal dialysis; that is, a dialysis covered by the thin internal wall (fig. 4). As incomplete retinal dialyses are not rare, some authors hold that most cases of retinal dialysis in the young are formed in this way. If a retinocele gives rise to a retinal detachment, operation is indicated; the prognosis is generally good owing to the peripheral location of the breaks. Massive vitreous retraction, however, has been observed in such cases.

The differential diagnosis between a primary detachment and a detachment associated with a choroidal tumor can be made in most cases without great difficulty, as a solid choroidal mass is generally seen under the elevated retina. Certain tumors, however, are associated with a serous retinal detachment and such cases may create great diagnostic problems. The following points may be helpful in arriving at the correct diagnosis (fig. 5).

1. In case of tumor, the detachment very rarely shows a retinal break. If retinal folds are present, they move slowly as though the fluid in which they float were viscid.

2. The detached retina is often adherent to the most protruding portion of the tumor, which is mottled with pigment; it is more grayish than a normal choroid and frequently shows small hemorrhages on its surface. The protruding part of the tumor may be hidden by overhanging folds of retina, or it may be flat and resemble closely a patch of old chorioretinitis. Often in such cases the diagnosis cannot be made by ordinary ophthalmoscopy. When no adhesion between tumor and retina is visible, a solid mass may sometimes be detected behind the detached retina in the subretinal space. It is advisable to use a strong light for this purpose. When monocular ophthalmoscopy is used, slit-like illumination will prove useful, but stereopsis with slit-like illumination is a far better method of detection. Indirect illumination often proves useful in making the dark outline of the tumor appear more clearly.

3. The existence of a choroidal mass can often be detected by transcleral or transcorneal transillumination. A subchoroidal collection of blood or a benign choroidal melanoma cannot, however, be distinguished from a malignant choroidal tumor by this method.

4. If the tumor is in the upper part of the fundus, serous fluid may sink in the lower half, causing a transparent detachment with retinal folds in this area. When the patient sits up, there may be two apparently unconnected detachments in the fundus; one above, due to the tumor itself, and one below, due to serous fluid. As the patient lies down, the serous fluid shifts upwards and collects around the choroidal tumor. In any case where two apparently unconnected balloons of detachment are observed—one above, which is unaffected by the patient's position, and one below, which moves upward when the patient lies down—a choroidal tumor should be suspected.

5. Usually, in case of choroidal tumor, the vitreous is not degenerated and vitreous detachment is absent.

6. A careful search for metastases in other organs may be of assistance.

SUMMARY

In many cases, the early symptoms of retinal detachment are: localized flashes of light, often below and nasally; a sud-

den increase in vitreous floaters; and, not infrequently, a vitreous hemorrhage of moderate amount, which may obscure fundus details. When the detachment extends, a gray cloud appears in the visual field, and the light flashes disappear.

Between 40 and 60 per cent of all detachments occur in myopes and, in cases seen by the writer, over 20 per cent were in aphakic patients. Slit-lamp examination may reveal atrophy of the posterior pigment layer of the iris, which often indicates marked degeneration of the extreme fundus periphery. In the deep portions of the vitreous, a detachment of the posterior hyaloid may be found, which appears as a tense membrane stretched across the vitreous cavity. In practically all cases of retinal detachment, a field defect may be detected provided one uses small test objects or reduced illumination.

Ophthalmoscopy. If a direct ophthalmoscope is used for fundus examination, it should have a strong illumination and a wide range of minus lenses. For examination of the anterior portion of the fundus, the writer uses a binocular indirect headband ophthalmoscope combined with scleral indentation. Ophthalmoscopy should supply information on the following points:

Is the retina detached? The common signs of retinal detachment are described. When the diagnosis is doubtful, stereopsis, strong light for direct and indirect illumination, observation of parallactic displacements, and indentation of the sclera should be employed. These methods also help to detect retinal breaks.

2. Extent of the detachment. In fairly recent cases, this serves as a rough guide to determine the location of the retinal breaks (fig. 21). A demarcation line indicates that the detachment has been present for over three months and has remained fairly stationary.

3. Location of the retinal breaks. The characteristics of a retinal break are described. Diagnosis may be difficult because of hazy media, flat detachment, or choroidal atrophy. Most retinal breaks are found either in the equatorial region or near the ora serrata. Equatorial breaks are most often located in the upper temporal quadrant; they are either tears or holes occurring in degenerated areas. Breaks near the ora serrata cause a detachment which, as a rule, progresses slowly. Usually they are of one of the following three types:

a. Small and multiple, forming either tiny dialyses or breaks related to meridional folds. These often occur in patients over 45 years of age and may be associated with quiescent peripheral uveitis, posterior cortical cataract, and deep chamber glaucoma. This is the type of retinal break most frequently observed in aphakic eyes with retinal detachment.

b. Single retinal dialysis occurring in young patients, predominantly affecting the lower temporal quadrant and having a tendency to be bilateral.

c. After a direct nonperforating eye injury, there may be multiple traumatic retinal dialyses that are often invisible unless marked indentation of the sclera is used. Concomitant avulsion of the base of the vitreous is frequent.

4. Status of the macula. If detached, the macula seldom recovers a normal visual acuity. The differential diagnosis between a macular cyst and a macular break is helped by the use of slit-like illumination and stereopsis. The presence of a macular operculum and the existence of a central detachment which is not explained by other visible breaks point toward the diagnosis of macular break.

5. The status of the vitreoretinal adhesions is indicated by three types of fundus appearances, each one of which makes the prognosis poor. The first is

characterized by fixed folds which may be circular and generally located at the equator, or star-shaped and located either below the horizontal or near the macula. The second is massive vitreous retraction, in which all the retinal folds are fixed, most of them irradiating from the disc. This occurs most often in detachments of long standing, after a vitreous hemorrhage of moderate or large amounts, and after retinopexy. The third fundus change that markedly influences the prognosis is the presence of equatorial breaks with rolled-up convex edges.

Differential diagnosis. A retinocele appears as a limited elevation of smooth and thin retina, showing obliterated vessels, glittering spots like snowflakes and larger reflections that are sometimes visible through an undilated pupil. The resulting field defect has steep limits; usually no retinal breaks are present, and the lesion is often nonprogressive.

In case of choroidal tumor, there are no retinal breaks; the tumor has a grayish surface and mottled pigmentation with small hemorrhages. The tumor often forms a dark mass which is visible behind the detached retina and may be adherent to it. Other indications are the coexistence in some cases of an immobile upper balloon of detachment caused by tumor, and a readily mobile lower balloon caused by subretinal fluid; the absence of vitreous degeneration; and the presence of metastases in other organs.

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LEGENDS FOR COLOR PLATES

Figure 1. Early detachment with vitreous hemorrhage.

Clinical history. Woman, age 49, with some myopic astigmatism; uncomplicated intracapsular cataract extraction in the right eye. Three weeks later, temporal photopsia with sudden dimming of vision in the same eye. On examination, blood was found in the vitreous and under the hyaloid. After a few days the vitreous became clearer as the blood settled along the ora serrata below. The origin of the hemorrhage was traced back to a large retinal tear in the upper temporal quadrant. Surface diathermy was applied over the tear and a double line of surface diathermy was made in order to connect the region of the tear with the ora serrata at 8:30 and 10:30 o'clock. The immediate postoperative course was uneventful; five weeks later, corrected vision was 20/50 in spite of some vitreous haze. The later course is described under Figure 15.

Fundus picture. In the upper temporal quadrant: retinal tear bridged by obliterated retinal vessel and surrounded by a minimal detachment; several foci of old chorioretinitis between equator and ora serrata. In the inferior part of the fundus: subhyaloid hemorrhage; blood in the vitreous, near the ora serrata. (M.B., R. S. 674)

Figure 2. Retinal detachment with retinal cyst.

Clinical history. Fifty-nine-year-old emmetropic man with an upper temporal detachment of the right eye. Good visual result after two operations. Four months after the second operation, recurrence with new retinal break in the upper nasal quadrant, but the patient refused further surgery.

Preoperative fundus picture. Two horseshoe-shaped tears at 10 and 11 o'clock, oval hole at 9 o'clock, and retinal tear at 6 o'clock, in an area where the retina was in situ. In the lower temporal quadrant, retinal cyst with several round holes and obliterated retinal vessels in its internal wall. In the lower nasal quadrant, several circumscribed areas of quiescent chorioretinitis. Extreme periphery invisible on account of lens opacities. (M.B., R. S. 103)

Figure 3. Optical section of eyes observed with a slit lamp and a strongly concave lens in front of the patient's eye.

(A) Superoposterior vitreous detachment limited by a wavy grayish line that is well visible on a dark background but practically invisible on the red background of the fundus. It is the writer's opinion that this grayish line is probably not the hyaloid membrane. A similar but less marked grayish line is visible below and immediately behind the lens.

(B) Aphakic eye of a 70-year-old woman, who had been emmetropic; an uncomplicated intracapsular cataract extraction was performed in 1944 with a good visual result. In June 1947, she had a retinal detachment operated on without success. In November 1947, a scleral resection was performed with a good result and vision improved from counting fingers at 2 feet to 20/200. The other eye was blind. Three and a half years later, sudden dimming of vision, suggestive of a recurrence of detachment. Ophthalmoscopic examination revealed no fundus change except for a marked turbidity of the vitreous.

Slit lamp picture. Hazy vitreous containing many pigment granules; in approximately the middle of the vitreous cavity, clearly visible membrane on which many grayish and pigmented granules were deposited. This membrane was stretched across the whole vitreous cavity and was adherent to the retina in the equatorial region, producing a sharply outlined circular retinal fold all along the equator. Fortunately, the chorioretinal scar of the previous retinopexies was located between the circular retinal fold and the disc, thus preventing the retina from becoming detached more posteriorly. When first seen, this membrane, which is assumed to be the posterior hyaloid, was so dense that the fundus could hardly be seen with the slit lamp. Later, it became more transparent, and vision returned to its previous level of 20/200. Posterior to the membrane, the vitreous cavity appeared optically empty. (F.S., R. S. 3)

Figure 4. Detachment with demarcation line.

Clinical history. Emmetropic woman, age 23, with poor vision in the left eye for one year. Extensive detachment operated on twice, with improvement of vision from counting fingers at 1 foot to 20/200.

Fundus picture. Fairly flat detachment except below, and complete demarcation line bordering its upper margin. In the lower temporal quadrant, retinal cyst extending over the pars plana, with obliterated retinal vessels and three

irregular breaks in its internal wall. On the convex border of the cyst, tufts of vitreous haze pointing towards the center of the vitreous cavity; the external wall of the cyst had been torn at the ora serrata and was retracted near its convex border, thus forming an incomplete retinal dialysis. Other features: irregular retinal hole at 6 o'clock, small tear with operculum at 9:30 o'clock, macular cyst, scattered retinal hemorrhages, and pigmentary degeneration of the extreme temporal periphery.

In small circle, pupillary reflex, which was gray in the area corresponding to the retinal detachment. (V.R., R. S. 527)

Figure 5. Retinal detachment due to a choroidal tumor.

Clinical history. Woman, age 71, with failing vision in the right eye for one year. Previous diagnosis: primary retinal detachment. Present diagnosis: detachment secondary to choroidal tumor. Enucleation. Pathological report (from Department of Eye Pathology, Massachusetts Eye and Ear Infirmary): solid intraocular tumor, 17 mm. in diameter and 4.5 mm. thick, in the upper nasal quadrant, reaching a region 2 to 3 mm. from the optic disc and macula. Microscopic examination: malignant melanoma of the choroid with extraocular extensions along posterior ciliary vessels.

Fundus picture. Detachment forming two balloons: an upper one always present regardless of patient's position, and a lower one that flattened out when the patient was lying down. The two balloons were smooth and connected with each other by an area of very flat detachment which might have escaped notice. No retinal breaks found. The upper balloon had been erroneously interpreted as a limited primary detachment surrounded by a demarcation line. When examined with a strong light and stereoscopic ophthalmoscopy, a choroidal mass of dark gray color, irregularly pigmented, and with a few hemorrhages on its surface could be seen behind upper balloon of retinal detachment. In one small area of the upper balloon, near its inferior border, the tumor was adherent to the detached retina. In the region where the tumor was visible, transillumination was poor. (M.K., R. S. 972)

Figure 6. Bilateral dialyses in the young.

Clinical history. Emmetropic man, age 21, with blurred vision in the left eye for one week before examination and no complaint in the right eye. A bilateral retinal dialysis was found, and both eyes were operated on with a good result. Vision in the right eye remained normal, and in the left, it improved from 20/400 to 20/70.

Fundus picture. In the lower temporal quadrant of the right eye, incomplete retinal dialysis containing three complete holes; minimal retinal detachment. In the left eye, inferior detachment, with a retinal dialysis at 6 o'clock. (D.T., R. S. 1086)

Figure 7. Retinal detachment with typical lesions of the region of the ora serrata.

Clinical history. Emmetropic man, age 57, with upper detachment in the left eye, probably of one week's duration. Operation was successful with vision improving from 20/200 to 20/30.

Fundus picture. Numerous meridional folds above—especially above and nasally; small dialysis at 1:30 and 2:30 o'clock; two small round holes at the posterior extremity of a meridional fold at 11:30 o'clock, and one round hole near the ora serrata at 2:30 o'clock. Advanced pigmentary degeneration all around the region of the ora serrata, particularly below. (W. McK., R. S. 528)

Figure 8. Typical retinal tears.

Ophthalmoscopic fields (inverted images) near the equator, in the upper half of the fundus. Left: retinal tear in an area of chorioretinal degeneration; an operculum floats in the vitreous to the right of the tear; obliterated vessel near the border of the detached area. Right: typical horseshoe-shaped tear with retinal flap attached to the anterior border of the tear, and retinal vessel bridging it.

Figure 9. Multiple dialyses of traumatic origin.

Clinical history. Emmetropic man, age 18, was struck with a finger in the right eye twelve months before examination; vision poor since that time. Total detachment operated on successfully.

Fundus picture. Total fairly flat detachment. Six dialyses visible: small at 10 o'clock, large from 10 to 12:30 o'clock, one between 2 and 3 o'clock, and three small dialyses between 5 and 6 o'clock. Except for the dialysis between 2 and 3 o'clock, they were invisible without indenting the globe very markedly, as they were either very small or flat and slit-like. A whitish ribbon hung in the vitreous

at the extreme periphery of the fundus above and another below. Each ribbon was attached to the ora serrata and pars plana at both ends. Clumps of pigment on the ribbons corresponded to ora teeth. These ribbons were probably the vitreous base which was pulled off the ora serrata and pars plana by the blow. As a result of the traumatic avulsion of the vitreous base, flat dialyses were produced and, from 12:30 to 2:30 o'clock and from 4:30 to 5:30 o'clock, abnormal pigmentation of the ora serrata and pars plana had occurred. The ciliary epithelium, over the pars plana, was detached in the upper temporal and in the lower nasal quadrant. (K.P., R. S. 1402)

Figure 10. Scleral indentation in the equatorial region.

Case of flat retinal detachment where no breaks could be found owing mostly to vitreous haze.

Ophthalmoscopic fields (inverted images) of the equatorial region, in the upper half of the fundus. Left: no scleral indentation; the retina is detached and appears thinned in front of an area of quiescent chorioretinitis. Right: scleral indentation makes the retina appear much more gray: two obvious holes are seen on the edge of the degenerated area. (A.K., R. S. 495)

Figure 11. Macular lesions observed with the slit lamp and a highly concave lens in front of the patient's eye. Fundus illuminated from the right side.

A *and* B: *Clinical history.* Emmetropic woman, age 54, with spontaneous temporal detachment of two weeks' duration in the right eye. Large horseshoe-shaped tear near the equator at 9 o'clock; detached macula with vision reduced to counting fingers at one and a half feet, and macular cyst. Reattachment followed application of surface diathermy over the equatorial break. One month later, a real macular hole had formed and was surrounded by a limited and very flat detachment, but in spite of it vision had improved from counting fingers at one and a half feet to 20/200.

Fundus pictures. (A). Macular cyst before operation. It was a little smaller than the disc and half filled with blood. Above the blood level, the thin internal wall of the cyst was visible only under slit-like illumination. On the left, the beam of light cast a hazy streak on the choroid. (B) Macular hole after operation. The internal wall of the cyst had ruptured, and blood had escaped. The defect had slightly rolled-up edges, and the beam of light cast a clearly outlined streak on the choroid. A complete macular hole was present, causing a flat perimacular detachment. (A.B., R. S. 795)

(C) *Clinical history.* Emmetropic man, age 20, with a spontaneous detachment of the left eye, described under figure 22 D, and a cystic macula.

Fundus picture. The thin internal wall of the macular cyst was visible only under slit-like illumination. On the left, the beam of light cast a hazy streak on the choroid. The outline of the cyst was irregular and there was a suggestion of three smaller cysts below the large one. (L.C., R. S. 855)

(D) *Clinical history.* A boy, age 12, was hit in the right eye with a baseball bat three years ago; poor vision for at least one year. Temporal detachment with several small breaks in the upper temporal quadrant near the equator, and a macular tear. After diathermy was applied over all the breaks, including the macular tear, the retina became reattached and central vision improved from counting fingers at 2 feet to 20/100.

Fundus picture. The macular tear was about the size of the disc and an operculum was clearly visible on the right. The edges of the tear were sharply outlined and slightly rolled up. The beam of light cast a clear-cut streak on the choroid, at the bottom of the tear. (C.C., R. S. 1188)

Figure 12. Circular fixed fold and breaks with rolled-up edges.

Clinical history. Moderately myopic woman, age 46, with limited detachment of approximately three weeks' duration, in right eye. A retinopexy was performed with good visual improvement.

Fundus picture. Row of equatorial retinal breaks from 9:30 to 11 o'clock, three of them having a rolled-up convex edge. Between 9 and 10 o'clock, two thin areas, also bordered by a rolled-up edge, were probably breaks in the process of formation. Several other breaks near the ora serrata between 11 and 12 o'clock. From 9 to 10:30 o'clock, circular fixed fold on the anterior border of the breaks. Tufts of vitreous haze, pointing toward the center of the globe, were attached to the fold. Obliterated vessels at 11:30 near the ora serrata; small retinal hemorrhage near the equator at 9:30 (A.C., R. S. 1380)

Figure 13. Retinal detachment with star-shaped folds.

Clinical history. Emmetropic man, age 58, with spontaneous retinal detachment of nine months' duration, and two star-shaped folds. After retinopexy, vision improved from counting fingers at 3 feet to 20/80. The star-shaped folds flattened out completely but, even twelve months after the operation, there was still some vitreous haze in the region of their former location.

Fundus picture. Subtotal detachment with one horseshoe-shaped tear near the equator at 11 o'clock. Two small star-shaped fixed folds in the inferior part of the fundus made the prognosis rather poor. Other features: several circumscribed areas of old chorioretinitis near the ora between 4 and 8 o'clock, meridional fold at 2 o'clock, and small hemorrhages at the posterior end of this fold. (B.L., R. S. 1020)

Figure 14. Normal ora serrata seen by indirect ophthalmoscopy and indentation of the sclera in a middle-aged subject (inverted images).

Left: normal ora serrata nasally. Deep scallops and marked ora teeth, bordered by a narrow pigmented band; minute meridional elevations, over the ora teeth and slightly anterior to them are beginnings of meridional folds. The uniform grayish color of the retina near the ora is not a sign of retinal detachment. Anteriorly, the pars plana appears dark, and a few ciliary processes are visible. Indentation creates a shadow that appears as a narrow dark band bordering the indented area posteriorly.

Right: normal ora serrata temporally. The anterior limit of the retina forms almost a straight line; the peripheral retina itself has a granular appearance owing probably to cystoid degeneration. The pigmented band is broad, it encroaches on the pars plana and its anterior border is scalloped.

Figure 15. Massive vitreous retraction.

Clinical history. (See figure 1.) Six weeks after retinopexy, the patient's vision suddenly dropped to hand movements at 3 feet. Fundus examination showed massive vitreous retraction. A scleral resection was performed without improvement.

Fundus picture. Typical massive vitreous retraction: numerous fixed folds radiating from the region of the disc; circular fixed fold with many whitish peaks protruding into the vitreous, along the equator below; total detachment except for the treated area. The location of the original tear was easy to recognize because its flap was still protruding into the vitreous in the middle of the treated area, and an obliterated retinal vessel was connected with it. New retinal breaks near the treated area at 9 and 10 o'clock. The subhyaloid hemorrhage had largely disappeared except for three small clots and for blood along the circular fixed fold. Blood, which had settled near the ora serrata below, appeared unchanged. (H.B., R. S. 674)

Figure 16. Typical retinal holes.

Ophthalmoscopic fields (inverted images) near the equator in the upper half of the fundus. Left: detached retina with five holes of various sizes in degenerated area. Right: detached retina with six holes of various sizes in area affected by lattice-like degeneration. This type of degeneration is characterized by white lines arranged as a lattice, and several of these lines are partly or completely obliterated retinal vessels.

Figure 17. Retinoschisis.

Clinical history. Emmetropic man, age 50, without complaints, was referred to the Retina Service with a diagnosis of retinal detachment in the lower temporal quadrant, right eye. On examination, a retinoschisis was found with similar but less advanced changes in the other eye. This patient was kept under observation for twelve months without visible changes.

Fundus picture. In the lower temporal quadrant, bulla with snowflakes and obliterated vessels; smaller bullous elevations in the upper temporal quadrant and at 6 o'clock; peripheral band of lesser elevation all around the periphery. Meridional folds in the upper nasal quadrant, on ora teeth and on the convex border of several ora bays. Tiny cysts along the ora serrata, temporally (ophthalmoscopic picture of cystoid degeneration). No retinal break found. (F.W.F., R. S. 1209)

Figure 18. Detachment at the ora serrata (inverted images).

Left: scleral indentation is applied slightly posterior to the ora serrata, and forms folds in the detached retina. They are particularly marked in the area of the shadow created by the indented choroid. Right: same region when the sclera is indented directly over the ora serrata, with shadow of the indented

choroid in the ora bays. Considerable increase of the apparent thickness of the retina (distance between the shadow and the upper limit of the gray retina), indicating that the retina is detached at the ora serrata and that the bays of the ora have been torn off their insertion to the choroid. The ciliary epithelium covering the ora bays is probably also detached from the choroid. The detached retina is granular, probably owing to cystoid degeneration.

Figure 19. Peripheral detachment in a high myope.

Clinical history. Woman, age 42, with 17 diopters of myopia in the right eye; vitreous hemorrhage of moderate amount, six weeks before first visit. When examined, blood in vitreous had largely absorbed but a smooth peripheral detachment was found between the equator and the ora serrata, in the upper half of the fundus. Retinopexy was followed three months later by a new retinal break and detachment on the disc side of the operative scar. After reoperation, a large vitreous hemorrhage occurred, followed by massive vitreous retraction and loss of useful vision. The left eye had similar lesions but was not operated on.

Preoperative fundus picture. Peripheral detachment walled off on the disc side by myopic chorioretinal degeneration along the equator. Retinal breaks at 9, 11, 2:30, and 3 o'clock. Obliterated retinal vessels in the detached area; peripapillary and macular chorioretinal atrophy. (M.M., R. S. 1124)

Figure 20. Diagnosis of a smooth, flat detachment.

Ophthalmoscopic field in the equatorial region (inverted image). In the upper part, no detachment; in the middle part, detachment causes fuzziness of the choroidal pattern and retinal vessels cast a visible shadow on the choroid. The lower part is not illuminated directly by the beam of the ophthalmoscope but by light reflected from the choroid. This indirect illumination renders readily visible the cobblestone appearance of the flatly detached retina.

Figure 21. Relationship between extent of detachment and location of retinal breaks, in relatively recent cases.

A detachment affecting one upper quadrant often shows a break in the superior part of the detachment (A).

A detachment affecting both upper quadrants often results from a break near 12 o'clock (B).

A detachment affecting one inferior quadrant (C and E) or both inferior quadrants (D and F) may be produced either by a break (generally a retinal dialysis) along the meridian bisecting the retinal detachment (C and D) or by a break in the upper portion of the detachment (E and F).

A ballooning inferior detachment is often the result of a break in one of the upper quadrants, usually the upper temporal (G); in such case detachment in the upper quadrant may be extremely flat and escape notice.

A total detachment generally results either from breaks near 12 o'clock (H) or from breaks in several meridians.

Figure 22. Demarcation lines.

(A) Emmetropic man, age 40, with blur in the right eye, one month before examination. Detachment with retinal dialysis in the lower temporal quadrant. Incomplete demarcation line, more marked at the periphery. Successful retinopexy with visual improvement from 6/200 to 20/40. (E.G., R. S. 931)

(B) Young girl, age 11, with moderate myopic astigmatism. Inferior detachment in the right eye with complete demarcation line involving the macula; small break in the 7 o'clock meridian. Successful retinopexy with resulting vision of 20/30. In other eye, similar detachment not operated on, with massive vitreous retraction and loss of all useful vision. (L.M., R. S. 573)

(C) Woman, age 30, with temporal detachment in the left eye, retinal dialysis in the lower temporal quadrant and complete demarcation line. After successful retinopexy, no appreciable visual improvement. (F.C.E., R. S. 117)

(D) Emmetropic man, age 20, with poor vision in the left eye for one year. Detachment with numerous small breaks near the ora serrata in the lower temporal quadrant. Several demarcation lines; one is almost complete except above the disc, and two others, in the lower nasal quadrant, are incomplete. Successful retinopexy without improvement of central vision owing to a large macular cyst, pictured in figure 11 C. (L.C., R. S. 855)

(E) Emmetropic boy, age 13, with a detachment of unknown duration in the right eye. Three small dialyses in the lower temporal quadrant and several incomplete more or less parallel demarcation lines near the edge of the detachment. Successful retinopexy with poor visual result owing to a macular cyst. (L.R., R. S. 1161)

(F) Emmetropic man, age 38, with a detachment of 16 years' duration in the right eye. It was total with breaks near the ora serrata at 10 and 2 o'clock, and several incomplete demarcation lines that had in the past been mistaken for foci of regular chorioretinitis. Successful retinopexy without visual improvement. In the other eye, detachment of 22 years' duration, with massive vitreous retraction. Deep chamber glaucoma in both eyes. (F.B., R. S. 660)

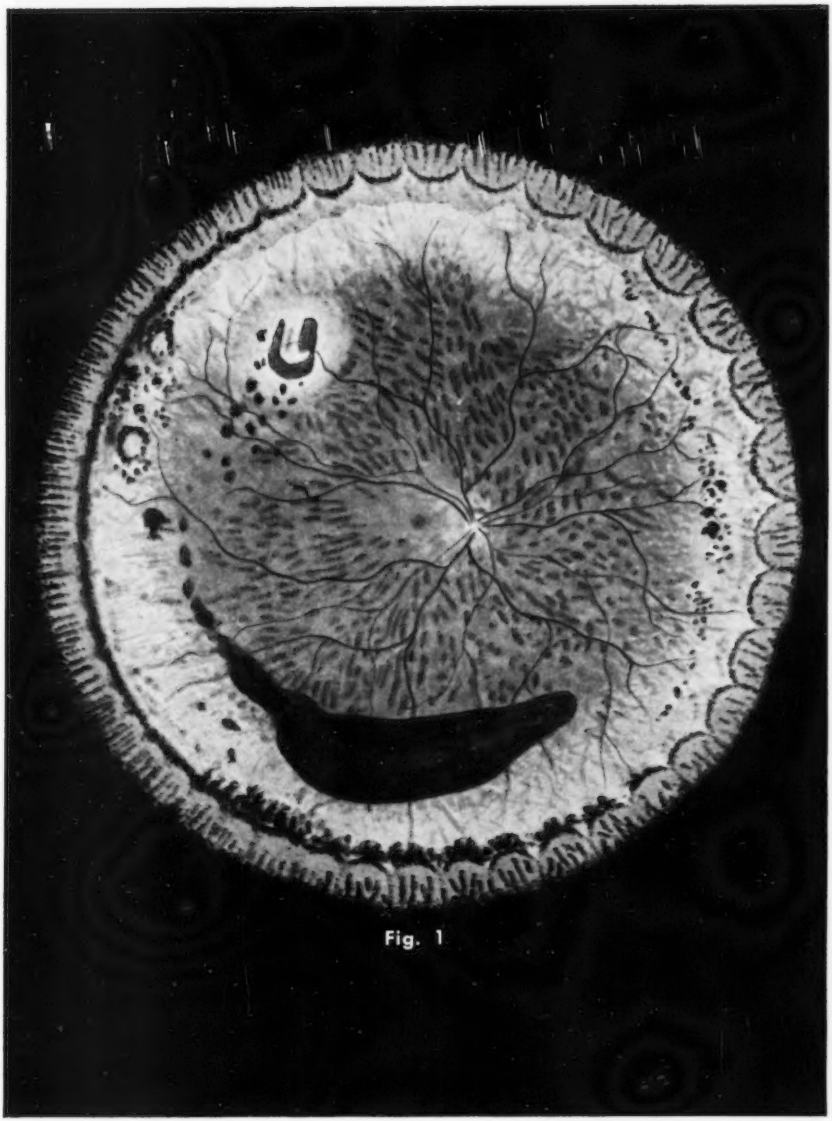


Fig. 1

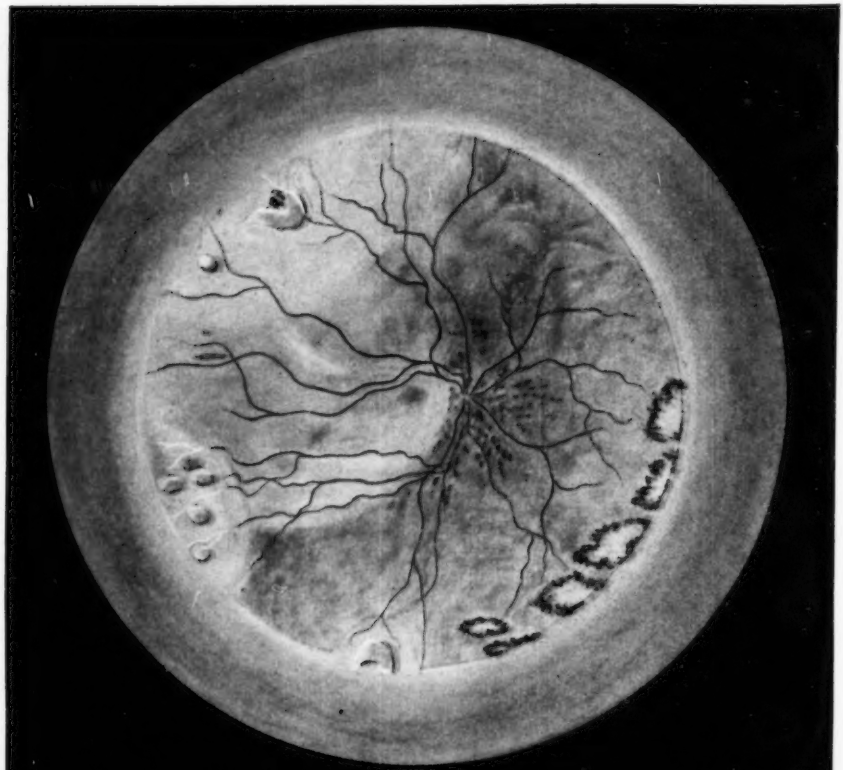


Fig. 2

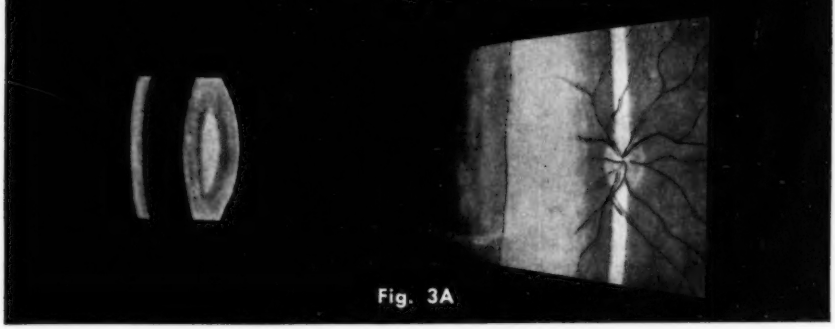


Fig. 3A

Fig. 4

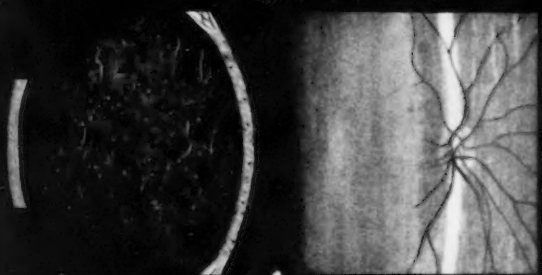


Fig. 3B

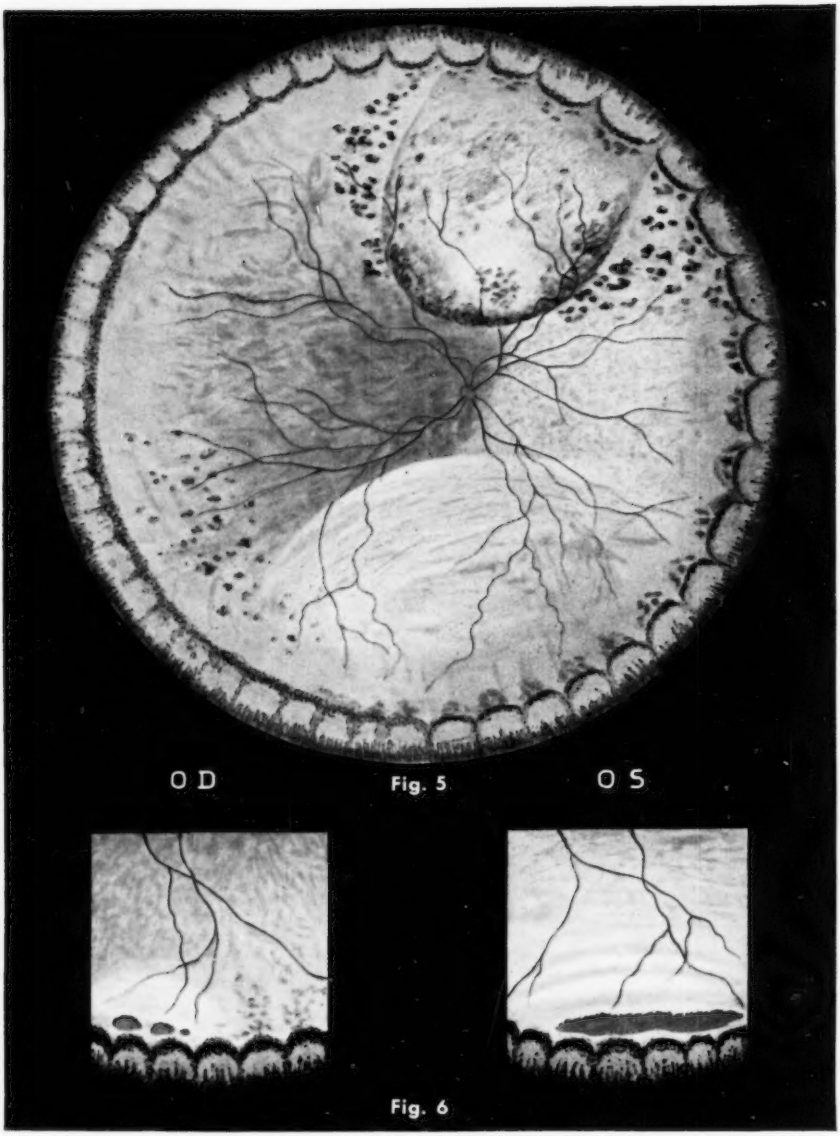
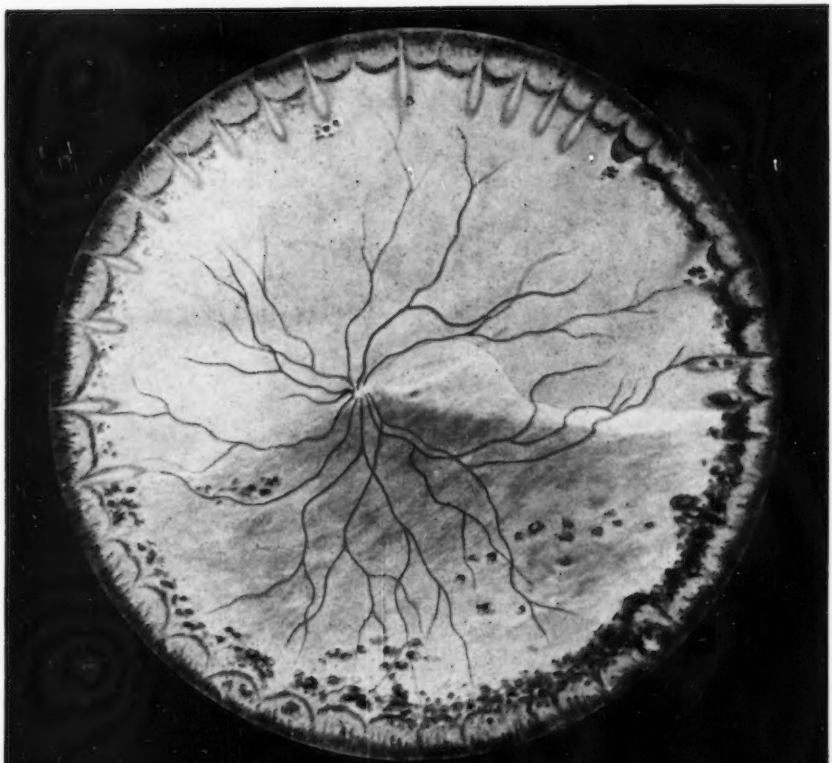


Fig. 5

Fig. 6



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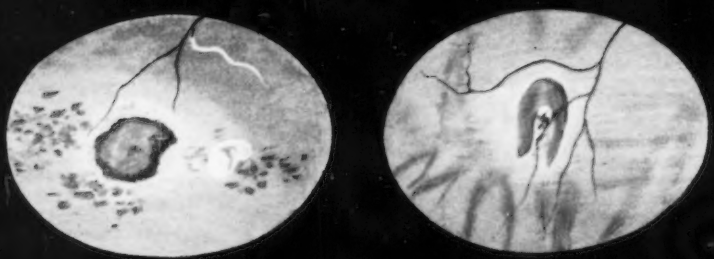

Fig. 8

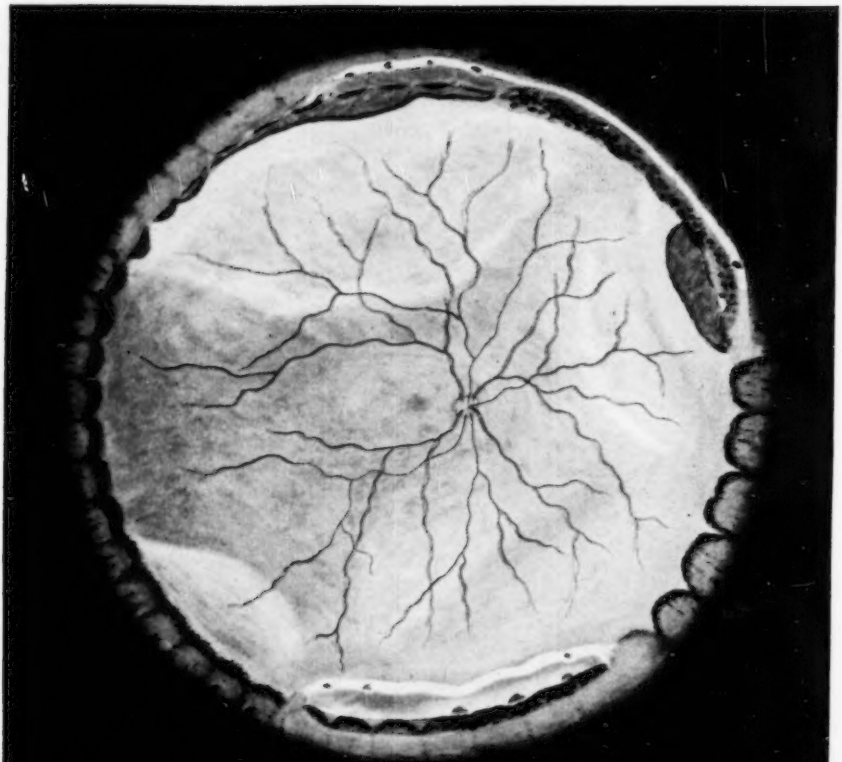

Fig. 9

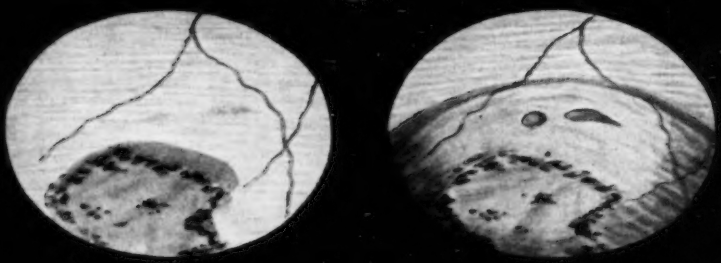

Fig. 10

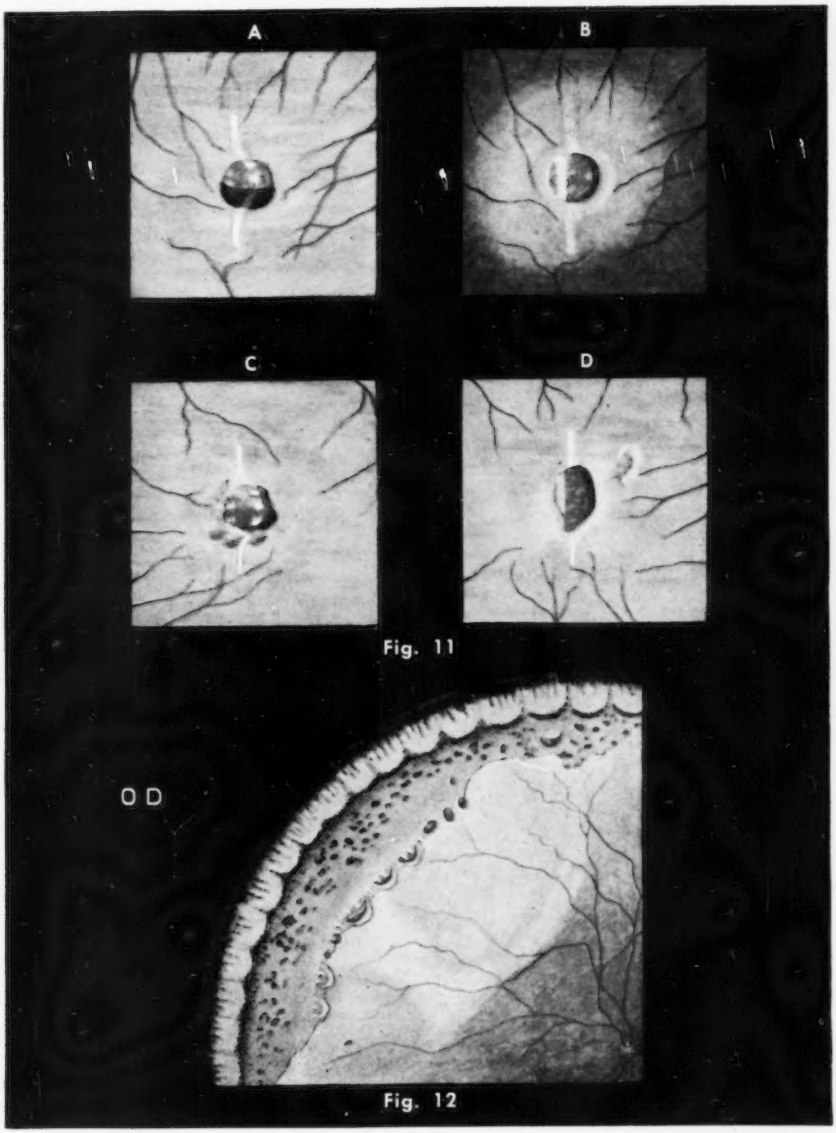

Fig. 11

Fig. 12

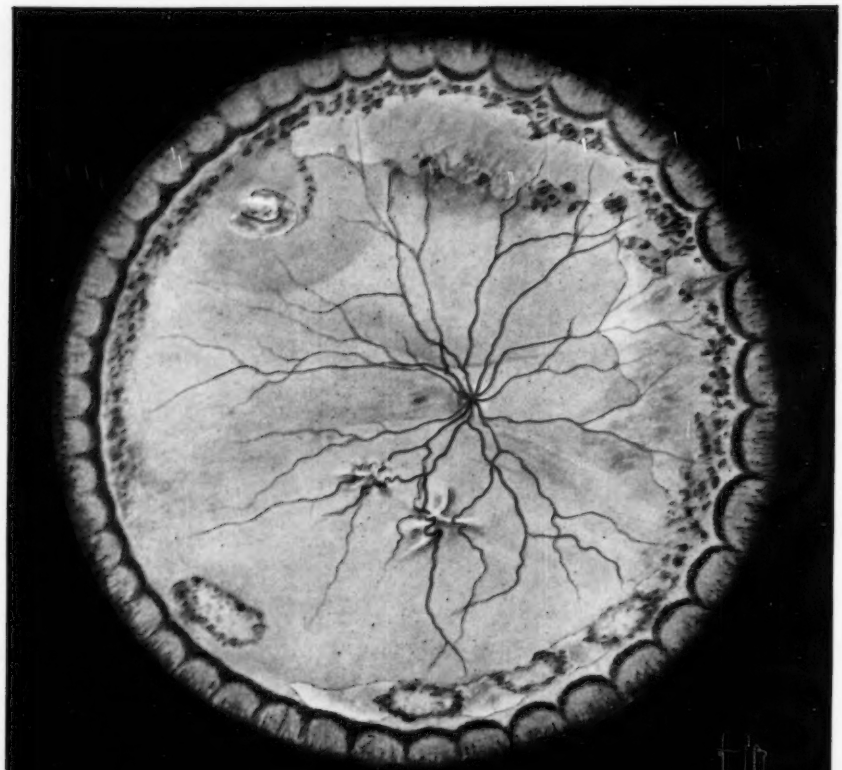

Fig. 13

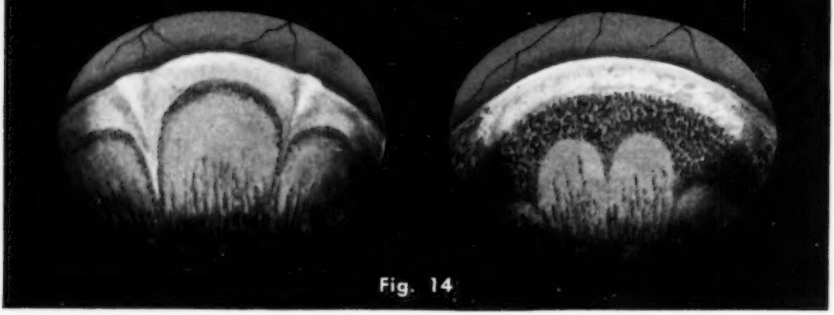

Fig. 14

Fig. 15

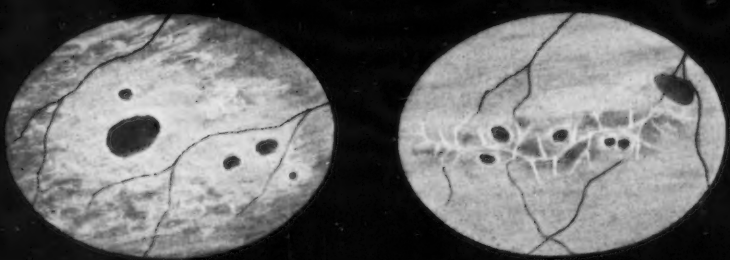

Fig. 16

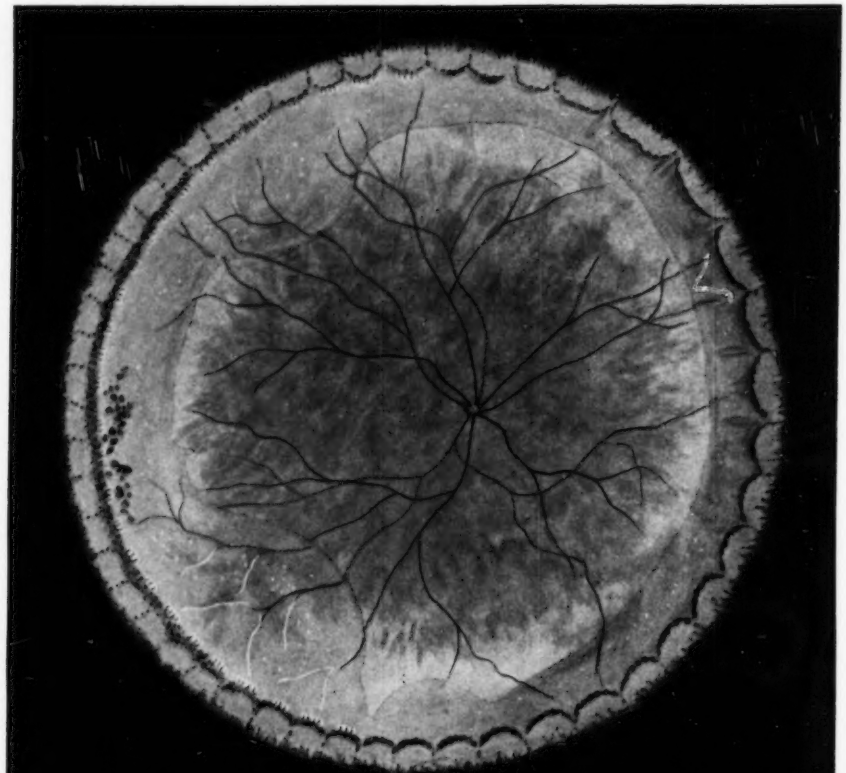

Fig. 17

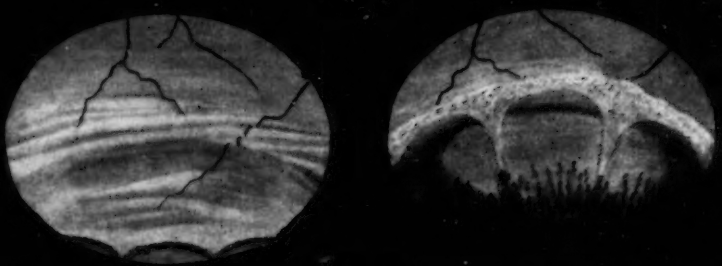

Fig. 18

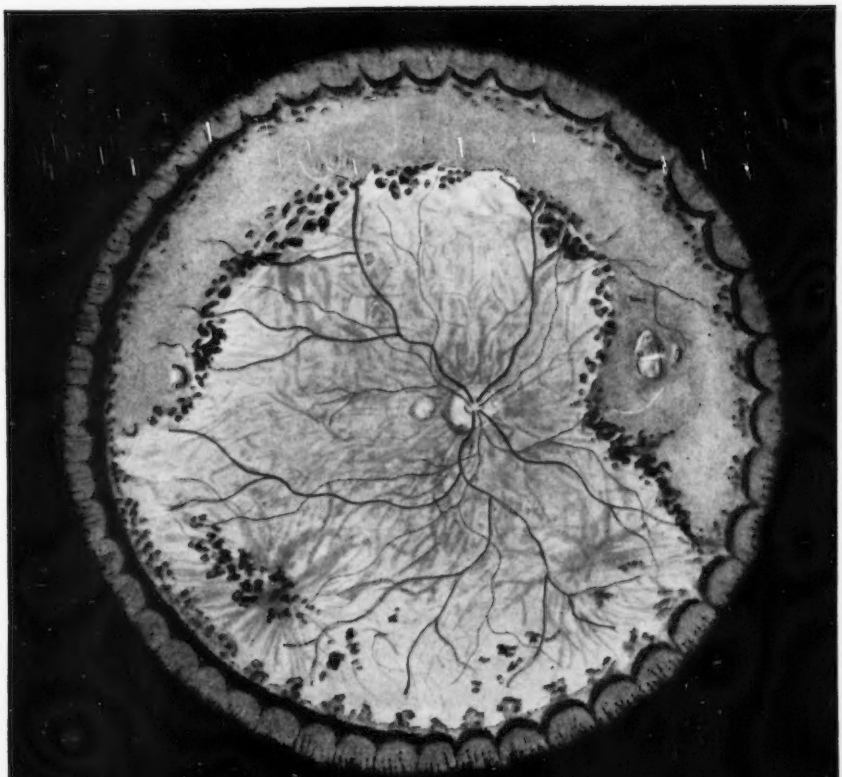

Fig. 19

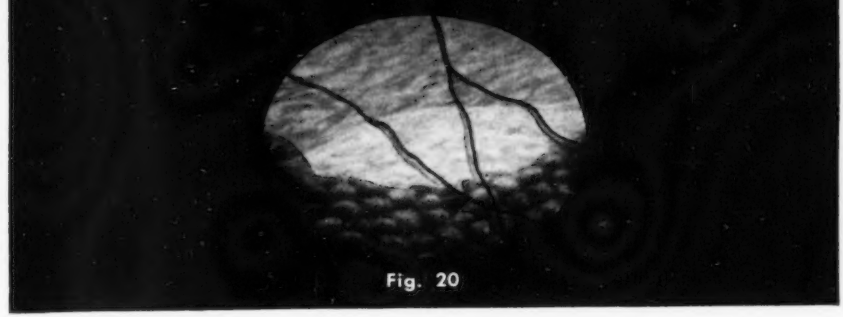

Fig. 20

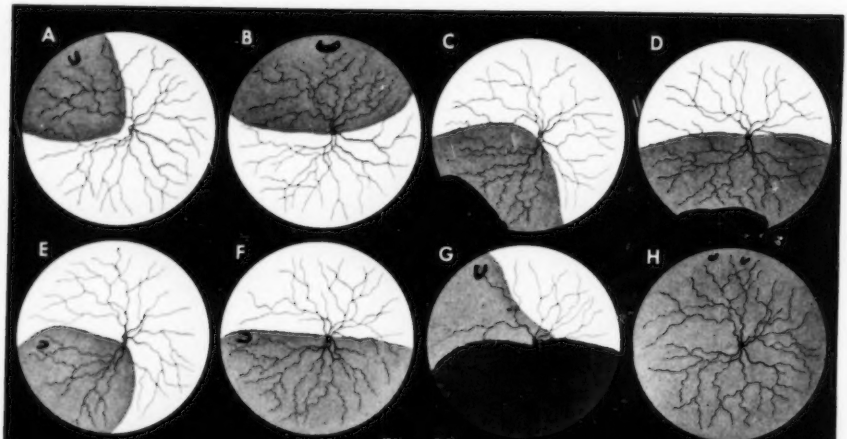

Fig. 21

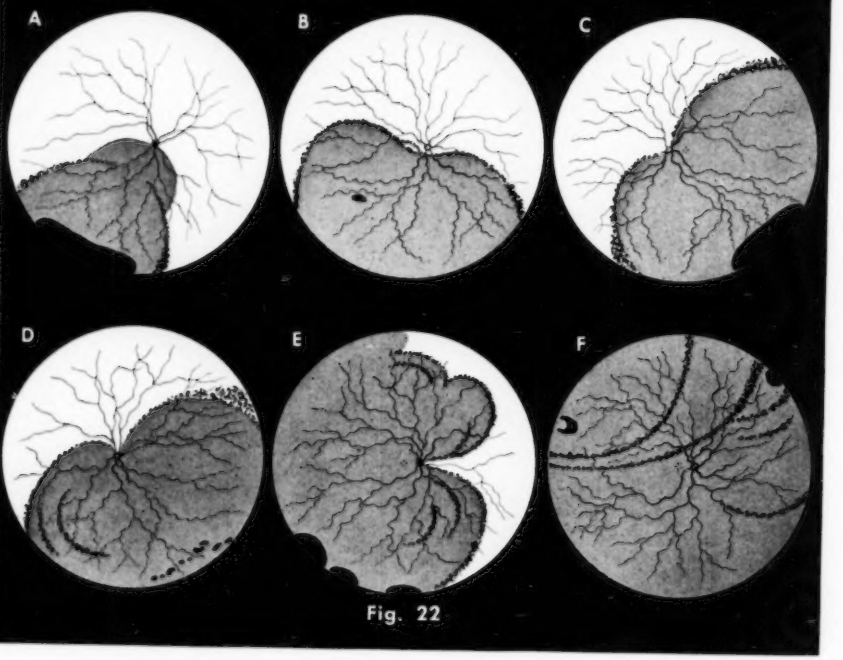

Fig. 22

TREATMENT OF RETINAL DETACHMENTS

Localization of Breaks, Description of a Typical Operative Procedure

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THE first step in the actual treatment of retinal detachments is the all important localization of all the retinal breaks so that an operation can be properly planned to close or obliterate them. It is important to localize the break or breaks at once because due to a settling out of the retina, they may disappear from sight on subsequent days.

The simplest way to localize the retinal breaks accurately is by means of a hand perimeter such as the Schweiger and a localization table such as developed by Stine.

The perimeter is held in front of the affected eye by the patient (fig. 1), and if possible, he fixes the center mark of the perimeter with this eye. If the vision of the affected eye is too poor for this, he must fix with the good eye. However, when this is done, in order to have the affected eye properly centered on the perimeter the patient must fix off center with the good eye. A simple device for accomplishing such fixation is a cotton-tipped applicator fastened to the perimeter so that the well eye will look far enough to one side to center the affected eye accurately on the perimeter hub.

The observer next finds the tear with the ophthalmoscope and then moves slowly away, while still staying focused on the tear by changing the lenses in the ophthalmoscope so as to keep the tear sharply in view. When he is just far enough away, the arm of the perimeter is swung around until it comes into the path of the beam of the ophthalmoscope light. Now two notations must be made at once, the meridian and the degrees of arc. The meridian is indicated on the hub of the perimeter, and this, if necessary, can easily be transferred to the eyeball at the time of operation. The degree of arc on the perimeter arm where the ophthalmoscope light strikes it must also be noted at once. Then with the aid of an appropriate table such as Stine's (table I), these degrees can be translated into the number of millimeters one must measure back from the limbus on the proper meridian to locate the hole accurately.

FIG. 1.—Localization with hand perimeter. This shows patient fixing off center with good eye, observer focused on tear, and arm of perimeter swung into beam of light from ophthalmoscope.

Department of Ophthalmology, Stanford University School of Medicine.

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TABLE I

TABLE FOR LOCALIZATION (Modified from Stine)

Degrees of arc:	90	85	80	75	70	65	60	55	50	45	40
Millimeters from limbus:	7.5	8.5	9.0	10.0	11.5	12.5	13.5	15.0	16.5	18.0	19.0
Degrees of arc:	35		30		25		20		15	10	5	0
Millimeters from limbus:	20.5		22.0		23.5		25.0		26.5	28.0	29.5	31.0

As soon as possible the patient should be put to bed with a binocular bandage for several days before the operation, to give the retina a chance to settle out. This is *very* important. As the bullous detachment unfolds, areas of retina previously invisible are seen and new holes may be found. Localization should again be made, as the hole may shift 10 or 15 degrees. With the retina nearly back in place, operation is much easier and more certain, as all operative steps can be followed more easily and with greater certainty. The importance of this assistance cannot be overemphasized.

The patient's head should be placed in such a position that the region of the principal retinal break or breaks is in as dependent a position as is practical. For example, with a break in the temporal part of the right eye the head is turned to the right. With a tear in the nasal side of the right eye, the head is turned to the left. For breaks in the upper half of the eye, the head should be reasonably flat, but a small pillow is allowable. Bell's phenomenon can be relied upon to turn the eye up far enough so that the upper half of the retina will be as dependent as is practically possible. Shock blocks under the foot of the bed are entirely unnecessary, as well as most uncomfortable for the patient. For breaks inferiorly, the patient may be raised to medium or high Fowler's position as seems indicated.

Sometimes the detachment practically disappears overnight and then, if the tears have not been localized, may be very hard to find. At other times the retina settles out slowly, day by day. Sometimes the detachment remains stationary for 48 hours, then suddenly recedes. At least 72 hours preoperative bed rest should be planned. However, if the retina has not receded in six or seven days, it probably would never recede by itself. But even many of these refractory cases can be cured.

This is perhaps the proper place to speak of the futility of so-called conservative treatment of retinal detachment. Unfortunately some men still try bed rest of many weeks, or compression bandages, or pilocarpine sweats, hoping that these conservative treatments may do some good. It is agreed by all workers that this is not only the sheerest waste of time but also jeopardizes the patient's chance for a successful cure through an early operation, for it is only by operative means that a simple detachment can be cured.

Taking up the operative treatment of retinal detachment, the question arises, "What operations can cure a retinal detachment, and how do they do it?"

Any operation that cures a retinal detachment does so by closing the retinal breaks. Five requirements must be met:

1. An exudative choroiditis must be produced.
2. It must be produced at the proper place.
3. The retina must be brought into contact with this choroiditis so that the edges of the hole or tear will touch this exudate.

4. It must be allowed to remain quietly in contact until a firm adhesion between retina and choroid is established.
5. This adhesion must be watertight.

Any operation that fulfills these requirements will cure a retinal detachment. Either actual cautery, chemicals, diathermy, electrolysis, cryothermy, or Shahan thermophore can be used.

How is a retinal break closed? Some of the older writers were of the impression that one cauterized the retina itself and thus sealed the break. This is not so. Any cautery or active electrode touching the retina will make a hole in it. This is frequently seen when long electrodes are used. Then this new hole must be closed by the exudate produced in the choroid by the electrode. This usually does occur as the retina settles back. But if the retina does not settle back, there is a new retinal hole to close at a subsequent operation.

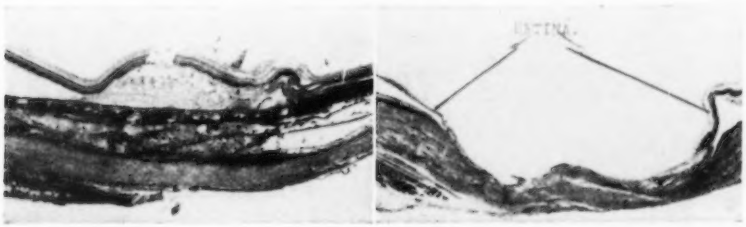

FIG. 2.—Closing retinal break (diagrammatic). (A) Histologic cross sections, showing exudate under retinal break. (B) Retinal break incorporated into atrophic healed scar.

The retinal break, or hole, or tear, whatever the name, is closed by incorporating it in a watertight retinochoroidal scar produced by the operative choroiditis (fig. 2, A and B). The preretinal fluid then can no longer get under the retina and lift it from its tenuous attachment to the choroid. Thus to cure a simple retinal detachment, each and every retinal break must be closed by a watertight retinochoroidal scar. The ideal operation for accomplishing this is one that can be accurately controlled, so as to set up fairly constant exudative choroiditis, and one that can be accurately localized and easily performed with simple apparatus.

The consensus of opinion at present is that the diathermy current is simplest, safest, and easiest to regulate as to dosage. Present-day diathermy apparatus is simple, reliable, inexpensive. The coagulating current is most commonly used and here the frequency should be between 750,000 and 1,500,000 per second. The cutting current is perhaps just as effective, but care must be used to prevent unnecessary destruction of tissue. In using the coagulating current, the milliamperes should be kept low, probably below 50 or 55 on any machine. The length of time the current is allowed to flow should be short, perhaps two to three seconds at most. These values will vary somewhat with different makes of machines. There will also be appreciable variations with the same machine in different localities because of the considerable variations in the actual voltage of the current in the hospital, even if it is rated as a 110-volt circuit.

The operator must learn to know his machine. In the final analysis, clinical experience is necessary to obtain uni-

formly consistent results. Here several points of importance should be mentioned. The most reliable criterion is the appearance of the retina after making a diathermy contact on the sclera. The appearance of the sclera is also helpful in evaluating the action of the current. The ease with which an electrode penetrates into the sclera is another helpful guide as to the proper amount of current.

For example, the speaker has found that on several different machines of the so-called Walker unit, manufactured by the Rose Company, 45 milliamperes of current applied for two seconds through a partially penetrating electrode will produce a very consistent retinal appearance and result in the production of sufficient exudate in almost all cases.

There are two points of view in operating. The first is to do as little as possible. The second is to do as much as the eye will safely tolerate.

To do as little as possible, one endeavors to close the tear or hole, and to treat nothing else. The advantage of minimal trauma is evident (plate I, fig. 1). The disadvantages are that one can miss a small adjacent hole or even part of a big hole, or leave untouched a degenerated area of retina which may tear later.

To do as much as possible one not only surrounds the hole itself but extends the barrage to include any degenerated area of retina or suspected retinal break; but one does not treat the whole area of the detachment.

With this second system one can treat an area extending more than half-way round the globe, thus including a number of breaks or degenerated areas (plate I, fig. 2).

It is not possible today to describe the considerable number of variations in technic employed by successful operators. They all have in common the fulfilling of the five conditions mentioned previously.

In the technic of diathermy operations there are three different ways of using diathermy electrodes. The first is by surface coagulation alone with a trephine opening through the sclera, or with one or two large or small diathermy punctures for drainage. The second is by use of perforating electrodes which provide many fine openings for drainage, a great advantage. The third is almost a combination of the two others, using special partially penetrating electrodes for surface coagulation instead of flat surface electrodes and also a number, 8 to 12, of perforating electrodes.

The advantages of the first technic, surface coagulation, are that the field is dry, the eyeball firm, and the application of the electrodes easy. One disadvantage is the uncertainty of transcleral dosage, and another is the difficulty encountered in draining subretinal fluid. If one large opening is made, as with a trephine or one large diathermy electrode, the sudden gush of subretinal fluid through this opening may sweep the retina into, and partly out of, this opening. If the retina ruptures, formed vitreous will escape. In such cases there are great possibilities of big hemorrhages and the development of retinitis proliferans with its bad consequences. Furthermore, the retina, drawn into the opening, will be put on the stretch and will develop stiff folds, pulling it from its base. Therefore, these large openings are to be condemned.

A much better way to obtain drainage with this method is to make one or more scratch incisions through the sclera, within the diathermy barrage, down to the choroid. The exposed choroid can then be carefully perforated with a fine diathermy needle. The subretinal fluid can then slowly escape through these openings. This technic obviates the great disadvantages just mentioned. However, the fact that sur-

face diathermy usually causes much more tissue necrosis and widespread atrophy of the retina and choroid still is a disadvantage.

The advantage of perforating diathermy, usually performed with one electrode, is easy application and very good drainage with little tissue damage or retinal atrophy. The disadvantages are that the eye gets soft, the field is wet, which makes for uncertain dosage, and the retina is often injured as it comes in contact with the long electrode as more and more of the subretinal fluid escapes. If a number of tiny electrodes or micropins are used, such as Safar, Walker and I have advocated, each being left in place until all are in, some of these disadvantages disappear, but the technical difficulties of putting in 20 to 40 pins are great. Furthermore, while the possibility of perforating the retina is much less, the danger of making some new holes remains.

The advantages of the third technic, with partially penetrating electrodes, are that the field is dry, the application easy, each one makes an easily seen mark on the sclera with each contact, thus, helping orientation, and finally, the 8 to 12 perforating electrodes give several different points for gradual and adequate drainage.

In this symposium the writer will describe only one operative routine which has proved satisfactory for a number of years. This technic uses partially penetrating electrodes for the major portion of the coagulation, combined with 8 to 12 perforating electrodes to provide drainage.

The advantages of the partially penetrating electrode over the plain flat surface electrode are several. The small point extending from its flat surface allows one to push the electrode far into the sclera. Then it cannot slide around, dissipating the current over an appreciable area of sclera. The point also carries the current through the sclera towards the choroid, which is where we need the current to insult the choroid so that it will produce the desired exudate. This electrode, once entered into the sclera, can be used almost as a tenaculum, to rotate the eye further to secure still better exposure.

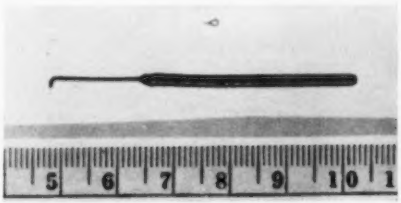

FIG. 3—Partially penetrating electrode with 1.0 mm. point; small Pischel pin for perforating diathermy.

For the above-mentioned operative routine, the Kronfeld electrode or the author's electrode, usually with the short 0.5 millimeter point, is used for the partially penetrating surface coagulation (fig. 3).

In some cases, as when the sclera is thickened by scar tissue, electrodes with longer points, such as 1.0 or 1.5 mm., may be needed for the partially penetrating applications.

When these are completed, Walker's micropins or the author's pins with 1.0 to 1.5 mm. points are needed to perforate the sclera. In some extreme cases a 2.0 to 2.5 mm. point may be necessary. Other special instruments include Lindner's localizing dial, Walker's rake ruler, the Arruga retractor, a curved scleral ruler, and thin and narrow insulated or plastic retractors. For macula holes, a self-illuminating plastic Arruga retractor (Berens) is helpful.

As to the actual operation itself, it can be performed satisfactorily under general or local anesthesia, although the writer prefers general anesthesia. For general anesthesia, avertin in full dos-

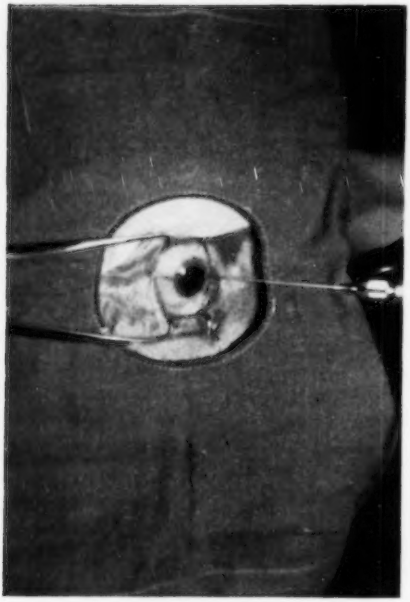

FIG. 4.—Marking horizontal meridian. This should be done at beginning of operation.

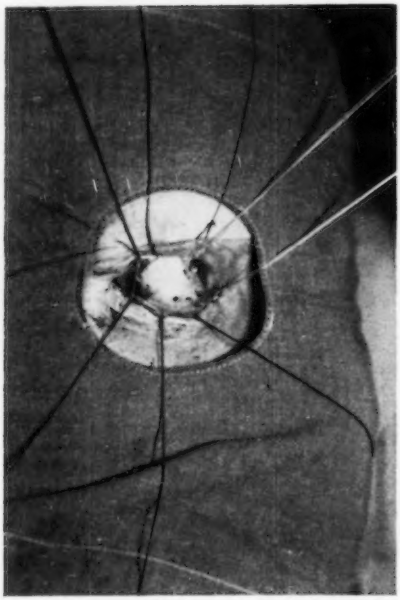

FIG. 5.—Good exposure of operative field. Note generous conjunctival incision with silk sutures holding conjunctiva free from operative field. Muscle has been tenotomized. White silk sutures in muscle stump and in neighboring muscle insertion. Lid suture in upper lid.

age, 80 to 100 mg. per kilogram of body weight is excellent. Intravenous pentothal sodium is preferred by others. Good medication an hour before the operation is necessary, such as dilaudid hydrochloride, 1/32 grain, or morphine, ¼ grain plus scopolamine, 1/200 to 1/100 grain. With the addition of local anesthesia, as described below, this is an excellent program.

For local anesthesia, preoperative medication as mentioned above together with nembutal, 3 grains, is satisfactory.

The pupil must be maximally dilated preoperatively to facilitate the frequent fundus inspections which are of utmost importance. Pontocaine is instilled for topical anesthesia. Then 2 per cent novocain is injected for akinesis, according to Van Lint. The horizontal meridian is marked at the limbus by a scratch with a needle dipped in gentian violet (fig. 4). Next a 1 to 2 cc. retrobulbar injection of novocain with epinephrine should be made. If considerable manipulation of muscles is expected, additional injection along the muscles is desirable.

For operation, a canthotomy is advisable in most cases. Next, a generous conjunctival incision is advantageous. The peripheral conjunctival edge should be secured with black silk sutures, which are then fastened with mosquito clamps and allowed to hang over cheek, forehead, etc. All this helps give excellent exposure. The sling sutures of white silk are now put around the exposed muscles and in the muscle insertion if a tenotomy was performed. Use *white* silk and *curved* mosquitoes to identify them and separate them from the black silk conjunctival sutures. If in doubt about adequate exposure, cut off a muscle after securing it with double-armed

suture. Do not hesitate to tenotomize a muscle. The great help this gives in exposure cannot be overemphasized (fig. 5). Rarely will troublesome heterophoria or heterotropia develop from this alone. Very troublesome heterophoria can come from the scar tissue and adhesions between muscle and sclera even without tenotomy. This is especially true after repeated operations.

scleral surface directly with the aid of the ophthalmoscope (fig. 6). For this an easy technic for using the ophthalmoscope is needed.* The assistant inserts an Arruga retractor or spoon over the approximate arc of the tear. The operator looks into the eye with a powerful ophthalmoscope and locates the break. The ophthalmoscope beam can be seen by the assistant as a surprisingly small

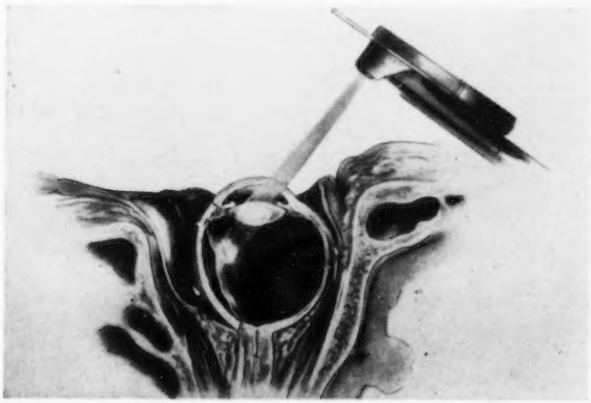

FIG. 6—Diagram of direct localization, showing Arruga retractor baring sclera, and light from ophthalmoscope showing on sclera.

The speculum can now be removed, affording still better exposure. Next a lid suture should be placed in either the upper or lower lid, whichever will be best, so that the lid can be kept over the cornea as much as possible. It is of great importance to keep the cornea covered so that it will remain clear.

After adequate exposure is assured, direct localization of each break is made. Direct localization means localizing the position of the retinal breaks on the spot of light on the sclera. He then marks this spot with the tip of an applicator dipped in gentian violet, thus localizing the tear accurately. If sterile caps and masks are worn, this maneuver is easily performed without contaminating the field (fig. 7).

Sometimes due to lens opacities or vitreous opacities, direct localization is not possible. Sometimes the cornea becomes hazy, again making direct localization impossible. This corneal haze may be due to using cocaine instead of pontocaine, or to exposure during the operation. Covering the cornea with the lid by means of a lid suture, as mentioned above, is the best method not only of correcting this condition but of avoiding corneal exposure. If the cornea

*The best ophthalmoscope for this is one like the American Optical Giantscope. It can be satisfactorily draped with an ordinary cord cover, to whose end a considerably wider hood has been sewn. This hood hangs back over the cord cover so that when the ophthalmoscope has been threaded through it, the head protrudes beyond the hood. With the hand grasping the encased scope, the hood covers the gloved hand to protect it from contamination by the user's face (figs. 8 & 9).

FIG. 7—Direct localization. Assistant marking sclera with applicator dipped in gentian violet.

must be exposed for any period of time, constant dripping of saline solution upon it will help maintain its clarity. The various solutions recommended in the literature for use when the cornea clouds up do not seem to help clear the cornea permanently for repeated fundus observations.

If, due to one of the above reasons, direct localization is impossible, use is made of the data obtained with the perimeter for indirect localization. The meridian can be estimated on the eye. It is better to use the Lindner localizing dial. The pointer is set at the proper meridian, the dial held on the eye over the cornea, with great care being taken to get the horizontal marks on the dial to coincide with the gentian violet marks (fig. 4) which mark the horizontal meridian on the eye. The meridian can thus be very accurately marked. The distance from the limbus in millimeters, also obtained from the perimeter and the Stine table, is measured on the proper meridian, and the tear is thus localized with great accuracy.

With the location of the retinal break marked, coagulation of the choroid is begun.

With some types of diathermy machines it is well, just before starting to apply the diathermy for the first time, to turn on the machine with the foot switch for a considerable number of seconds in order to warm up the tubes in the machine.

The placement of the diathermy applications is of utmost importance, for success in an operation depends on the precision of placement of the coagulating points. And precision can only be obtained with adequate exposure. Therefore, it is well to state again that a generous conjunctival incision is essential and that temporary tenotomy of one muscle should be done without hesitation.

Another reason for good exposure is that only in this way can vortex veins be easily seen and scrupulously avoided. Coagulation or even rupture of the extrascleral portion of a vortex vein is no calamity. The hemorrhage resulting from a rupture may be troublesome, but it can be stopped by adequate coagulation of the end of the vein. The great danger is in coagulating the ampulla of a vortex vein, which can easily happen with poor exposure. A still greater hazard is that of putting a perforating electrode through the ampulla. In either case, a marked hemorrhage into the eye may result, which usually results in a complete failure for the operation. Many of the massive intraocular hemorrhages reported by some ophthalmolo-

LEGENDS FOR COLOR PLATE I

Figure 1—Minimal treatment. The exudate just includes the retinal tear, and in healing, the choroiditic scar was just barely large enough to seal the tear.

Figure 2—Healing diathermy barrage, surrounding three retinal breaks, and walling of the affected area of retina.

Figure 3—Fundus view after three diathermy applications. Note three whitish spots, central to retinal tear. One has black center because too much current was used.

Figure 4—Fundus view with pilot pin. Partially penetrating applications do not show up, but perforating pin has touched retina.

Figure 5—Fundus view. Double barrage completed.

Figure 6—Fundus view. Double barrage completed and circle of perforating pins in place around tear.

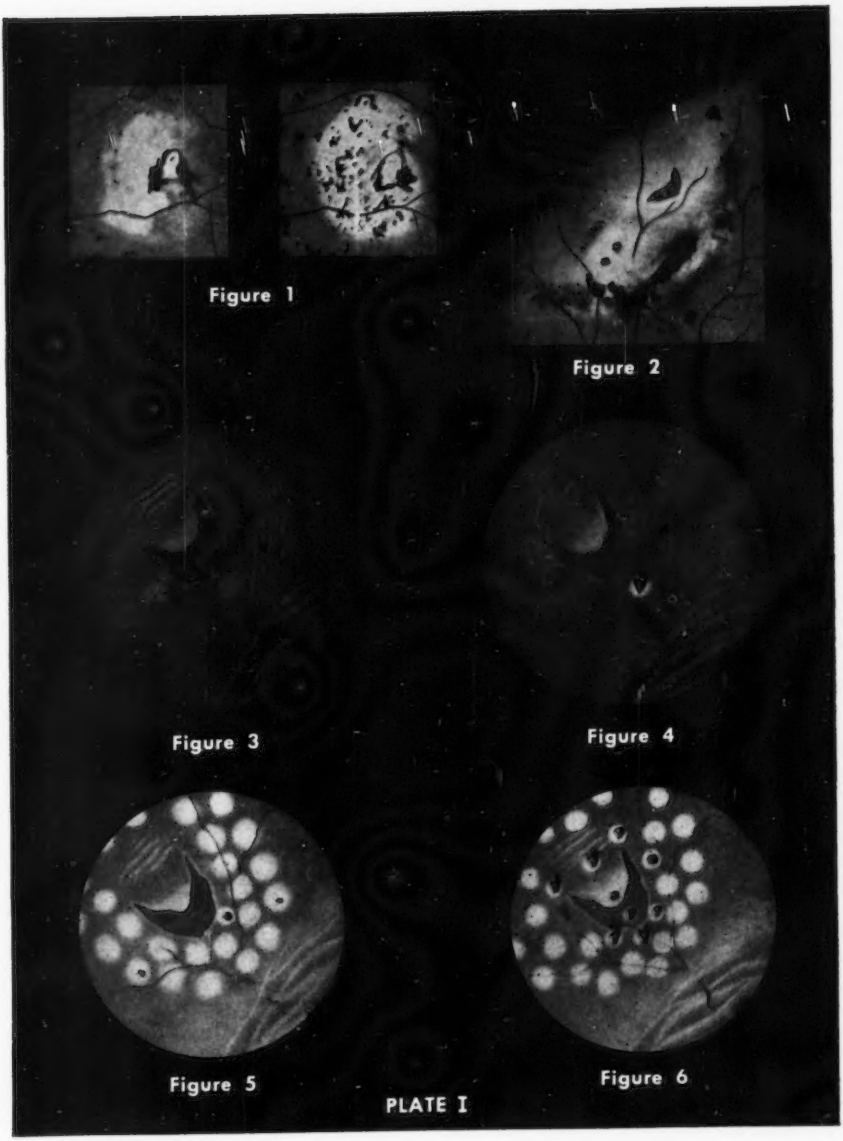

Figure 1

Figure 2

Figure 3

Figure 4

Figure 5

Figure 6

PLATE I

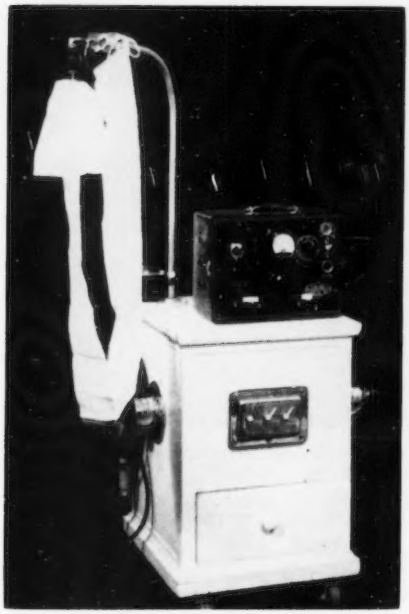

FIG. 8—Draped ophthalmoscope. This is hung from bracket on switch box, which holds diathermy machine. Switches on box control operating lights and ophthalmoscope.

FIG. 9—Technic for using ophthalmoscope in sterile manner. Note how gloved hand is protected.

gists are due to this avoidable accident. Strict adherence to the rule of no coagulation over a vortex vein or its ampulla has made massive intraocular hemorrhages very rare in our cases.

Conjunctiva and Tenon's capsule are retracted with nonmetallic Kronfeld retractors. The field should be wiped dry, preferably with applicators wrung out of distilled water. Two or three applications are now made just back of the tear with the partially penetrating electrode, spacing them 2 or 3 millimeters apart (fig. 10). It is not necessary, and perhaps is even harmful, to make the applications very close together. To do so causes increased necrosis of the sclera and widespread atrophy of the choroid and retina. They should be spaced with great regularity, however, to avoid any break in the ensuing barrage. Current below 50 to 55 milliamperes for two or three seconds is almost always sufficient to produce proper coagulation, but as stated previously, clinical experience will dictate the proper amount. Enough current and sufficient time should be used to get good reaction, but no more than necessary should be used. Overtreatment should be scrupulously avoided. Many eyes are lost because the operator in his uncertainty decided that if a little current was good, a lot more current was better.

After the first two or three applications have been made, the fundus is inspected. If the retina is only slightly elevated, these coagulations will show up in the fundus as round white areas in the retina, looking almost like "cotton-wool patches." If one sees them, one can tell whether the coagulation is being done in the correct place or not (plate I, fig. 3). If correct, further coagulation is completed.

If it was not possible to see the coagulated areas around the tear at the be-

ginning of the coagulating, a perforating Pischel pin or Walker pin can be put in over the tear as a "pilot pin." This almost always can be seen and allows for proper orientation (plate I, fig. 4).

FIG. 10—Diathermy aplications on sclera. Three applications have been made. The mark localizing the retinal hole on the sclera is just anterior to the lowest application.

If the pin has not made a white spot in the retina, it can usually be seen as a glistening object behind the translucent retina, and thus its position is accurately localized in relation to the fundus details. If even this fails, "reverse localization" can be done. To do this the irreplaceable Arruga retractor is used as before when the break was localized, but now the assistant tells the operator who is looking into the fundus when the ophthalmoscope light is on the pin. Thus the operator can localize the position of the pin in relation to the fundus details and orient himself properly. In the rare cases in which even this cannot be done, the location of the applications will have to be done blindly, according to the data obtained from the localization found with the perimeter.

When the operator is satisfactorily oriented, further coagulation is done, placing the new applications so as to surround the breaks or tears and any additional area of retina indicated. The applications are spaced regularly and carefully 2 to 3 millimeters apart. A double row of applications makes the most satisfactory and safest barrage (fig. 11). One wants to be sure to go *back* far enough to get well *behind* the tear. *Frequent fundus inspections are an invaluable guide* to proper placing of

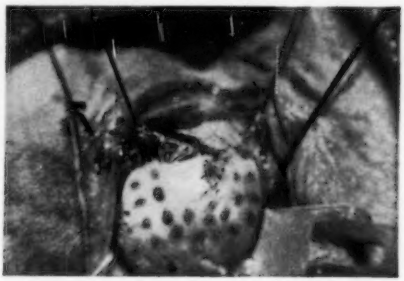

FIG. 11—Double barrage of partially penetrating electrode almost complete.

the barrage. This point cannot be overemphasized. Look, look, look! This is very important.

If the retina is elevated 4 diopters or less, the coagulations show up as white areas in the retina almost like large cotton-wool patches. Sometimes they have a tiny black spot in the center, which probably is the retina burned by too much diathermy current. Too much diathermy may also cause the formation of bubbles. The visible coagulated spots should form a regular, evenly spaced barrage. If some are very faint, more current may be applied. Where the retina is considerably elevated, the applications may not show up in the fundus. It may be necessary to measure on the sclera the distance of the applications from the limbus to be sure one has gone back far enough to place the applications properly. The barrage should also be carried forward to the ora serrata, which is about 6 to 7 millimeters from the limbus.

If one of the applications accidentally perforates and drains, an isolated pin, such as the Walker or Pischel, can be put in to act as a plug. This usually stops

FIG. 12—Putting in perforating Pischel pin, within barrage.

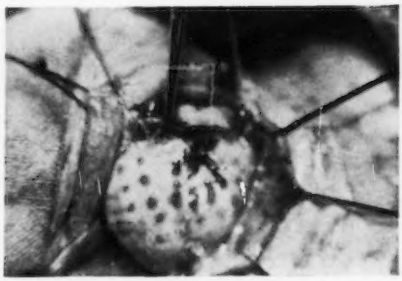

FIG. 13—Circle of pins around retinal break.

the leakage and keeps the field dry but does not hold very tightly, thus requiring care in further manipulations of the globe.

After the barrage is completed, 8 to 12 perforating applications should be made (fig. 12). The Walker pin or Pischel pin can be used for this. The pins should be placed around the tear *within* the nonperforating barrage and not outside of it (fig. 13). This is very important for the following reason: If a perforating electrode touches or perforates the retina, a new hole is made in the retina. This must be sealed by the exudate from the diathermy; and the amount of exudate is of course greatest within the barrage, so that the chances of closing such a newly produced hole are best in this area.

There is another reason for putting the perforating diathermy punctures around the retinal breaks. It is well established that the perforating diathermy electrode produces the firmest scar. The union between retina and choroid (and sclera) resulting from the perforating applications is much stronger than that produced by nonperforating diathermy applications. So it is wise to have the strongest adhesions around the retinal break where the continued pull of vitreoretinal adhesions is most likely to occur.

It is perhaps best not to place any pins directly over a large break. If the retina settles down so that such a perforation is not covered by any retina, a semipermanent fistula may result, with ensuing acute hypotony and its bad results.

It is not necessary to go far beyond the nonperforating barrage to get under the area of highest retinal elevation in order to obtain drainage from this point. The whole subretinal space is connected, so that the subretinal fluid from any location can flow towards the perforations made around the retinal break and drain out there.

Now the area directly over the retinal break should be treated with nonperforating diathermy, and if room exists, applications should be made over the base of a horseshoe tear.

An all important fundus examination should now be made, to be sure the retinal breaks are well surrounded by the diathermy coagulations, and to be sure there are no gaps in the barrage (plate I, figs. 5 and 6). If the retina had settled out well preoperatively, almost all the diathermy applications will show up well in the fundus and one can easily see whether the barrage is properly placed. If the retina is still quite elevated, or becomes more so during the manipulations of the operation (a frequent occurrence), some diathermy

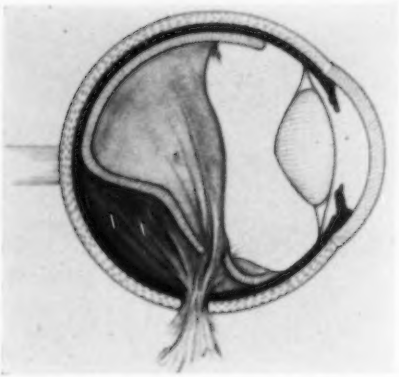

FIG. 14.—Diagram of faulty action of large trephine opening through sclera. Fluid rushing out through trephine opening.

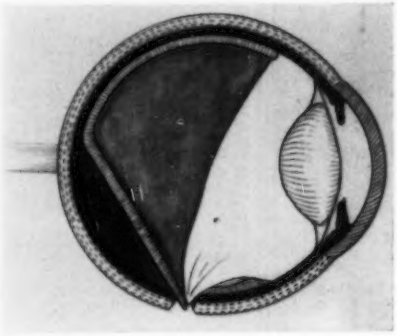

FIG. 15.—Retina and vitreous caught in large trephine opening.

points will not show up. This frequently is the case around horseshoe tears which are being held up by the pull of the vitreous. In such cases the operator must judge the location of the barrage by the position of the perforating pins as seen in the fundus. If the pins are seen at the tear, and on the sclera the nonperforating barrage is well posterior to the pins, all is well. If not, further coagulation must be done further posteriorly. At this time also any doubtful areas are further coagulated.

If all is well, the perforating pins are now withdrawn. Usually most, but not all, of the holes drain. If additional drainage is desired, fluid may be aspirated through these openings with an eye dropper.

If no drainage occurs, as sometimes happens, what is the explanation? The sclera may be exceptionally thick so that the pins have not perforated. Perhaps the subretinal fluid may be coagulated, specially around holes, or the subretinal fluid is too thick to drain easily. Or the holes are plugged in some other manner, or we don't know why.

What should be done? The answer to this question is variable. Nothing need be done if the retina is quite flat or if it settled out well preoperatively.

If the retina is still markedly elevated, additional diathermy punctures with a single longer electrode or with long pins may be made. The cutting current may be useful in getting a bigger opening to obtain further drainage.

Another way to obtain drainage, according to Schepens, is to make within the barrage one or more small incisions through the sclera, as in a cyclodialysis. The exposed choroid can then be directly punctured with a very fine diathermy needle. As a last resort, as stated by McLean, a careful trephine opening just through the sclera may be justifiable. This should be within the barrage, or a small additional loop may be made within which the trephine opening is set. After the scleral plug is loosened, the exposed choroid should be carefully perforated with a fine discission needle, making a tiny hole, to evacuate the subretinal fluid slowly. But one should not make this opening large or far back of the barrage. To do so would encourage the disasters seen so frequently with this technic. The sudden rush of fluid to the single large hole drags the retina, and often vitreous, with it, *into* the opening. The retina heals in here with resulting tension folds which prevent the retina

from settling back smoothly into its proper place, and another failure is recorded (figs. 14 and 15).

If with good drainage the retina still does not settle back, one may consider injections into the vitreous cavity. Air or saline can be injected into the vitreous cavity. A circle of diathermy points is made close to the ora serrata at a point where one is sure the retina is attached. A no. 28 or no. 30 needle in a scrupulously clean hypodermic syringe is placed on the sclera 10 or 11 millimeters from the limbus, and with diathermy current applied to the needle, it is easily pushed through the sclera. It is then pushed further into the vitreous cavity until it can be *seen through the pupil* by the assistant. Then 1 to 2 cubic centimeters of saline or air are injected slowly. Good drainage for the subretinal fluid must be present, as otherwise the globe might be ruptured by the tremendous increase in intraocular tension.

The great advantage of saline is that fundus details are still perfectly visible so that one can observe whether the retina is settling out. Its disadvantage is that it will run through a large retinal tear and that it is not compressible to provide pressure for several hours after the operation.

When air is used, it can be introduced under pressure so that for several hours it continues to press the retina back against the choroid. It is seen in the fundus as a large bubble, or more often as many small ones looking like frog spawn, coming just behind the lens, which naturally prevents any further view of the fundus. It absorbs within five to seven days, when fundus examination is again possible.

Injecting air into the anterior chamber is wrong. In phakic eyes nothing is gained by pushing the lens diaphragm back. In aphakics, air in the anterior chamber might cause glaucoma by slipping behind the iris and pushing it against the cornea.

Saline in the anterior chamber is free from the danger of inducing glaucoma. However, to produce pressure on the retina it must filter back through the lens zonules and push against the vitreous diaphragm. This is a most inefficient way to get pressure on the retina. Eventually the saline solution will filter back past the vitreous into the subvitreal space, so it might as well be injected in there in the first place if it is to do the most good.

Any tenotomized muscle is now resutured very loosely. Pulling it up tightly will produce overaction, as it will become very adherent to the sclera due to scar tissue over the diathermy-treated area. The conjunctiva is closed with a running untied silk suture. The canthotomy, if present, should be sutured and both eyes bandaged, and the patient is returned to bed. Now the postoperative treatment begins its important role.

Symposium: Retinal Detachment

POSTOPERATIVE CARE AND COMPLICATIONS

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THIS section deals with the events that occur in eyes affected with primary retinal detachment during the postoperative period. To a large extent these events are related to the operation; to a small extent they are continuations of the processes which make up the picture of primary retinal detachment and were discussed in previous sections of this symposium. The distinction between these two types of processes can be difficult but is essential for the evaluation of the effects of the surgical procedure. The average length of the postoperative period is about three months; the clinical recovery and total or partial disability period after successful detachment operations is somewhat shorter.

The multiform postoperative events occurring in eyes with primary retinal detachment will be discussed under three main headings: (1) the regular postoperative course; (2) irregularities in the postoperative course; and (3) complications.

A fairly sharp and obvious dividing line seems to exist between the regular and the various irregular courses after operation. The distinction between irregularities and complications made in this paper may be somewhat arbitrary.

From the Illinois Eye and Ear Infirmary, University of Illinois College of Medicine.
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THE REGULAR POSTOPERATIVE COURSE

The term "regular" as used here implies that the eye in question responds to the break-closing operation by a prompt return of the retina to the normal position. The fact of this reattachment is usually not established with certainty until the sixth, seventh or eighth postoperative day, at which time most operators make the first thorough ophthalmoscopic examination. Until that time, that is, during the first week following the operation, the risks entailed in ocular movements, the difficulties due to cloudiness of the media and sensitivity of the eye and the unsettled state of the retina make anything more than just a peek into the fundus inadvisable. During this first week the principle of the treatment is complete immobilization of both eyes in a position reducing vitreous traction on the area of the breaks to a minimum. Since in many cases of retinal detachment the topographic and mechanical relationship between vitreous and retina is not completely known (because of the limits of the present methods of examination), it has become the routine procedure to place the patient in a position which represents a sensible compromise between making the tears the lowermost portion of the retina and the patient's ability to maintain such a position. Avoidance of ocular movements is more important than the position of the patient's head. Sandbags are rarely indicated. The patient is advised not to pay any attention

to the noises around him and the hospital personnel is instructed in not making any noises that might induce reflex movements of the patient's eyes. The observation of this strict immobilization is not too hard on the patient, the hospital personnel or the visitors. In many respects the management of these patients does not differ from that of patients with cerebral concussion or skull fracture.

While his eyes should remain completely immobilized, the patient should receive every possible form of nursing care to the skin and muscles of his body which are taking the brunt of his weight. Systematic, active, arm and leg exercises, as well as breathing exercises, are started on the first postoperative day. These precautions, aiming at the prevention of pulmonary and circulatory complications, are most important in patients of the older age groups. The local treatment consists, again with a minimum of ocular movements on the part of the patient, of careful changes of the eye dressings every twenty-four to thirty-six hours and generous applications of drugs with long-lasting mydriatic and cycloplegic action.

At the end of the first week ophthalmoscopic examination is indicated, primarily to recognize those cases which have derived no benefit from the operation and should, therefore, be spared further useless discomfort and expense. If the postoperative course has been regular, the ophthalmoscopic visibility is usually found to be as good as preoperatively. In the area of the operation shiny white *intraretinal lesions* dominate the picture while the untreated retina is largely or completely in normal position. During the second week one observes the gradual transformation of the white, snowball-like areas of coagulation into chorioretinal scars. Around the tenth postoperative day the coagulated retina begins to become more transparent, exposing more and more of the reaction of the deeper structures to the coagulation. Spotty and streaky pigment proliferation interspersed with areas of choroidal atrophy dominates the picture from here on. The absorption of subretinal fluid in the untreated areas is often so fast that at the end of the first week the only elevated portion of the fundus is the coagulated area itself. This elevation is not due to a residual detachment but to flattening of the sclera and retinal and choroidal edema. The fresh coagulations, as a rule, form an effective though fragile barrier around the breaks. The observation of this settling-down of the retina associated with the formation of a wall of adhesions around the tears is one of the most gratifying, the most heartening events in ophthalmology, proving beyond doubt the correctness of Gonin's concept of the direct cause-and-effect relationship between retinal break and retinal separation.

The final appearance, shape and extent of the chorioretinal adhesions are dependent upon the type and intensity of the current as well as upon the shape of the electrode. Besides, the pre-existing condition of the choroid and the site of the application influence the final ophthalmoscopic appearance of the area of operation and its environs. These relationships make an interesting subject for detailed study but are beyond the scope of this symposium.

Once the watertightness of the wall around the break appears reasonably certain from the reattachment of the untreated area (specifically the area proximal to the barrage), the patient is given pinhole glasses and allowed to return gradually to an "up-and-around" existence. Pinholes that are two or three millimeters in diameter are as effective and much more practical than those of one millimeter. If the untreated portion of the retina remains in its normal posi-

tion, the patient is dismissed from the hospital with pinhole glasses some time between the fourteenth and twenty-first postoperative day. In working out the further program for these patients it should be remembered that the principal provocative agents, as far as the peace of the retina is concerned, are rapid, jerky ocular and head movements. Pinholes are usually worn until about eight weeks from the day of the operation. Visible transformation of coagulated tissue into scar tissue may continue during this entire period but should reach a standstill or completion at the end of the second month. If at that time the untreated retina is in normal position, the pinholes may be omitted for one or two days, after which the patient returns for another careful ophthalmoscopic examination.[3] If the latter proves satisfactory, the pinholes are kept off and the patient is allowed gradually to resume a normal life. Manual laborers as well as office workers are usually back at their regular work three months from the day of the operation. Abstention from violent physical exercises for another three months seems a wise rule for all patients. Continued close observation of the eyeground is indicated in borderline cases. Localized vitreoretinal adhesions in untreated areas of the fundus call for especially close observation and for continuation of the physical restrictions.

IRREGULARITIES IN THE POSTOPERATIVE COURSE

Among the kaleidoscopic variations of irregular postoperative behavior of retinal detachments, the following more or less typical courses may be recognized: (1) incomplete settling-down of the retina; (2) complete settling-down followed by recurrence; (3) complete settling-down followed by a detachment in a new portion of the eye ground; (4) no visible response to the operation, and (5) progression of the detachment.

By incomplete settling-down is understood a postoperative course characterized by a regression of the original detachment during the first week after the operation, but stubborn persistence thereafter of a part or parts of the original detachment. Such incomplete settling-down, in the writer's experience, usually means a favorable response of the retina to a well-planned and well-executed, but slightly imperfect, surgical procedure.

The most common cause of incomplete regression is a slight leak or gap in the barrier which gives rise to a small residual detachment. Such a gap may be purely accidental, and may occur in eyes operated upon under conditions of good visibility. More often, however, such gaps develop in eyes in which the operation was performed without adequate ophthalmoscopic control because of cloudiness of the media, and in cases of bullous detachment in which retina and choroid were not brought near each other during the operation. As long as the barrier forms well in all its other portions and as long as the residual detachment does not involve the central area or become progressive, a conservative attitude is permissible. A few of these barriers become complete by late extension of adjacent chorioretinal adhesions. If after a month of observation there are no ophthalmoscopic signs of closure of the gap, a second operation is indicated. If such a residual detachment should start to increase in area, a second operation should be undertaken without delay.

The first operation may prove inadequate because of a tear located outside the barrier produced by the first operation. The latter break may have escaped discovery before the operation or may have formed after the operation. The ophthalmoscopic visibility may improve as a result of the settling-down of the retina so that a break located on the

off-side of a bulla becomes visible. There again, nothing is gained by waiting longer than two weeks from the day of the detection of such additional tears. Early reoperations for either one of the situations mentioned in the foregoing may be difficult technically but are attended by a high percentage of permanent success.

Rare instances of residual circumscribed detachments after otherwise successful operations may be due to inability of the choroid to absorb the subretinal fluid. The fault may lie in the choroid itself, which in some cases of this type is suggested by ophthalmoscopic signs of choroidal atrophy, or in an unusual viscousness of the subretinal fluid. The dynamics of the fluid exchange between subretinal space and choroid have not been sufficiently unraveled to explain satisfactorily all cases of residual detachment. It is a fact, however, that some of these residual detachments — residual in the face of an apparently complete barrage around the breaks—subside completely and lastingly after surgical evacuation of the subretinal space.

In the irregular situations described so far, the postoperative course of the detachment was regressive at first and then became stationary. This course is primarily indicative of inadequacy of the surgical procedure and not of an unusual structural abnormality of the retina or vitreous. A second operation is likely to succeed, since the nonspreading of the detachment characterizes the detachment as fundamentally benign.

A very different situation is present if complete or almost complete settling-down is followed by a recurrence a few weeks later. In these cases the situation seemed encouraging during the first month after the operation in that the retina settled down and good choroidal adhesions were forming. The patient left the hospital very markedly improved and with high hopes for a permanent cure. A few weeks later the detachment recurred in the sector originally involved and surgically treated. New breaks just posterior to the surgical chorioretinal adhesions or an actual tearing away of the retina from these adhesions proves to be the cause of the recurrence. The appearance of the retina in several such cases observed by the writer has suggested intrinsic shrinkage or retraction of the retina which Arruga[1] has stressed as the chief pathogenetic factor in retinal detachment. The possibility of injurious effects of unnecessarily strong and extensive coagulations must also be considered. The prognosis in such cases is unfavorable. Some of them are benefitted by a scleral resection posterior to or in the area of the coagulations if such a procedure is feasible. Weve's reefing procedure[5] may be suited for some of these cases and is easier than the scleral resection.

Another typical variation in the postoperative course of retinal detachments is complete subsidence of the original detachment followed one to three months later by a new detachment in a new location. Some of these cases represent manifestations or continuations of the disease that caused the original retinal detachment. A typical example of this situation would be a case in which the original detachment was caused by a horseshoe-shaped break in the upper temporal quadrant. After several days of immobilization, the upper nasal quadrant of the retina proved to be in normal position but contained one or two atrophic spots with or without circumscribed vitreoretinal adhesions. The surgeon may have considered treating these atrophic spots at the time of the first operation but decided against doing so for technical or other reasons. The postoperative course was thoroughly regular until, about two months later, the atrophic spots could be seen to turn into breaks and to give rise to a new de-

tachment which was probably not related to the first operation. A second operation aiming at the new break or breaks has a good chance of being successful, assuming a state of soundness of the lower half of the retina.

In some of the cases of new detachments, especially if the new site is diametrically opposite to the original one, the mechanism seems to be different and a relationship to the operation is very likely. Breaks may or may not be found. A characteristic feature of such cases seems to be the presence of well-formed vitreous in the parts of the eye accessible to biomicroscopy with the fundus contact lens or a strong concave lens. The new detachment shows signs of intimate contact with contracting vitreous and does not respond to immobilization of the patient. The biomicroscopic findings especially suggest a relationship or physical connection between the two sites of detachment. The concept of vitreous traction originating from the area of the first operation seems plausible.[4] Such detachments are notoriously difficult to cure. Scleral resections combined with diathermy coagulations of breaks posterior to the resection seem to offer a better chance than the typical diathermy procedure.

Another unfavorable variation of the postoperative course is that of no visible effect of the operation upon the position of the retina. This course of events is usually not recognized with certainty until the seventh or eighth postoperative day. Assuming that the coagulations were made in the proper places, there are three possible mechanisms accounting for the stubborn maintenance of the status quo on the part of the retina. It may be physically unable to move toward the choroid (1) because of vitreous strands or bands holding it, or (2) because of an internal, truly retinal rigidity caused by a diffuse atrophying disease, or (3) because of inadequate drainage. If the evidence points in the direction of inadequate drainage, immediate reoperation aiming at evacuation of the subretinal space by means of the technics described by Dr. Pischel is indicated. The original coagulations, if not more than a week old, can still become effective if the retina is brought close to them.

A persistent detachment due to fixation of the retina by vitreous strands or by internal rigidity is very difficult to influence. Attempts at cutting vitreous strands are fraught with many difficulties and dangers and have been unsuccessful in the writer's hands while scleral resections have improved the relationship between the fixed retina and the choroid in some of the writer's cases.

The most alarming irregularity in the postoperative course is progression of the detachment. A relatively benign form of postoperative progression of the detachment is due to excessive electrocoagulation. This is particularly apt to happen in cases with multiple breaks and relatively little elevation. In trying to cover the edges of all breaks with ophthalmoscopically visible coagulations the operator may apply the current for longer periods than absolutely necessary. This overdose of the high frequency current leads to excessive heat in the subretinal space with marked expansion of all its contents, which results in greater elevation of the retina. As long as all breaks are covered by the coagulations, it has been the writer's experience that this progression is self-limited and that the outcome is favorable.

Serious postoperative progression occurs in an early form and also in a late one. The early form is characterized by the development, during the first few days after surgery, of tense rigid bullae separated by deep valleys with no or minimal signs of settling-down at the site of the operation. The visibility re-

mains sufficiently clear to reveal all the painful details of this postoperative course. It is highly probable that the main mechanical factor operative in these cases is massive retraction of the vitreous. To the writer it also seems probable that this vitreous retraction is related to one specific feature of the operation, namely a drainage opening allowing an entirely uninhibited and lasting flow of intraocular fluid out of the eye, called by Lindner[3] "free fistulation." These cases illustrate the dangers entailed in trephine or other large single drainage openings, especially if placed directly over large retinal breaks.

The late form of progression of the detachment occurs characteristically three to six weeks after an apparently successful and uncomplicated operation, the retina almost over night assuming the ominous umbrella shape. Again, massive vitreous retraction appears to be the underlying mechanism to which overly extensive and diffuse electrocoagulation may have contributed in some not completely understood manner. Scleral resections have been tried in both forms of massive vitreous retraction, with some success in the early and with hardly any success in the later form.

COMPLICATIONS

There are a number of true complications occurring during the postoperative period which are related to the operation or its effects upon the ocular tissues. A benign form of iridocyclitis follows extensive application of the current to any, but specifically to the anterior, portions of the sclera. Suppurative infections of the interior eye after electrocoagulations are extremely rare. A typical but fortunately infrequent complication is hemorrhage into the vitreous cavity or into or between the layers of the eyeball wall. The fact that after the operation parts of the retina tend to move toward the choroid while others are still held by vitreous entails physical stresses that can easily lead to retinal hemorrhages. Bleeding from choroidal vessels may be due to partial sloughing. Hemorrhages into the vitreous cavity may assume very bizarre shapes because of the complicated anatomic conditions prevailing in eyes with retinal detachments.

Massive retraction of the vitreous has been mentioned as a postoperative irregularity but probably represents a complication. The same status has to be accorded to the star-shaped retinal folds which occur postoperatively in different sizes and shapes due to several pathogenetic mechanisms. In some cases it is an intraretinal atrophying process,[2] whereas in others vitreoretinal adhesions or newly formed membranes on the inner retinal surface dominate the picture. While star-shaped folds unquestionably occur in eyes not operated upon, they are more common after operations and are, therefore, possibly a direct or indirect effect of the electrocoagulation. Weve's method[6] of treating such areas with roentgen therapy in the early stages and with electrocoagulation aiming at the foci of connective tissue in later stages has been unsuccessful in our hands. Scleral resection may be considered as a means of bringing the multiply folded retina closer to its matrix, the choroid.

The problem in the treatment of the postoperative irregularities and complications of retinal detachment is the question of whether, when and how to operate again. Just as during the preoperative work-up, close ophthalmoscopic study will prove most helpful in arriving at the proper decision.

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EVALUATION OF RESULTS OF RETINAL DETACHMENT SURGERY

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THIS part of the symposium concerns the statistical evaluation of the results of retinal detachment surgery. The material consists of 375 consecutive cases of primary retinal detachment in both phakic and aphakic eyes operated on by members of this symposium, the attending staff, or residents at their respective hospitals. Each case has been followed for at least six months after the last operation. After the selection of various preoperative, operative, and postoperative factors which were considered possibly important in the success or failure of retinal detachment surgery, these data were gathered by the participants and recorded on punch cards for analysis. Although the percentage of successful operations was lower in aphakics than in phakic eyes (table I), viz., 44 per cent versus 72 per cent, it was the opinion of the Committee, borne out by the analysis of the cases, that similar factors predisposing to success or failure were present in both groups, without assuming any influence of the presence or absence of a lens per se on the result of operation. The difference in frequency of these various preoperative conditions in phakics and aphakics will be described subsequently. Otherwise, both groups will be considered together, in which 67 per cent were cured by operation. No cases have been excluded except where information on a specific factor is lacking, and this accounts for different totals in the various tables. In accordance with the usual custom, no statistical correlation will be considered as significant unless the probability of error (P) is 5 per cent or less.

TABLE I

RESULTS OF OPERATIONS ON RETINAL DETACHMENT IN PHAKIC AND APHAKIC EYES

	NO. OF CASES	PERCENTAGE CURES AFTER OPERATION
Aphakics	64	44
Phakics	314	72
		P * = < 0.001

*P = probability of error

It is the purpose of this analysis to correlate various preoperative conditions of the eye, operative technics, and postoperative complications with (1) reattachment of the retina, and (2) visual results.

REATTACHMENT OF THE RETINA FOLLOWING OPERATION

This group includes those cases which obtained a reattachment of the retina or surgical "cure" without consideration of the resulting vision. A total of 102 recurrences or extensions of the detachment (27 per cent of 379 cases) developed after an apparently successful operation. The majority of these occurred within the first four months following operation (table II). The recurrence rate after a few months is undoubtedly

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TABLE II

TIME OF RECURRENCE OF DETACHMENT AFTER A SUCCESSFUL OPERATION

DURATION BEFORE RECURRENCE	NO. CASES FOLLOWED	PERCENTAGE RECURRENCE
1-2 months	379	18
3-4 months	288	5
5-6 months	257	2
7-11 months	229	1
1-2 years	159	6
3 years	79	1
4-5 years	27	4
6 years and over	12	8

TABLE III

REFRACTIVE ERROR (MYOPIA) VS. REATTACHMENT

REFRACTIVE ERROR	NO. CASES	PERCENTAGE CURES
Emmetropia or Hypertropia	171	78
Myopia, 1-3 D.	58	71
Myopia, 4-9 D.	60	65
Myopia, 10+ D.	16	50
"Near-sighted"	7	29

($P = < 0.01$)

too high, because fewer "cures" return for examination. Unless otherwise specified, "cure" will indicate that the retina has remained in essentially normal position for at least six months following the last operation, only 15 per cent of the recurrences occurring after this period. Included as cures are 34 cases where the retina was in normal position as seen by ordinary examination and the break was well surrounded by chorioretinal adhesions, but on more detailed examination by the method of Schepens, a slight elevation of perhaps one diopter was found within this area of barrage peripheral to the equator. Such a condition persists indefinitely and will be classified as a "cure."

Preoperative Conditions

The following conditions of the eye found on preoperative examination appeared to have some influence on the prognosis for reattachment after operation. Progressively increasing amounts of myopia were associated with a gradual decrease in operative cures (table III). The extent of the retinal detachment as determined by the number of quadrants of field defect (table IVa) was found to be directly related to a poorer prognosis either after one operation or after multiple operations. However, the extent of the detachment as seen by ophthalmoscopic examination showed a poorer prognosis only if the detachment was complete (table IVb), although the figure of 48 per cent cures in total detachments is rather surprisingly high. There was a suggestive but not statistically significant relationship between the degree or depth of the field cut and the percentage of cures following operation (table IVc).

Retinal breaks. The importance of detection and localization of breaks has been justly emphasized. In this series, the percentage of cures was highest in those cases where breaks were detected

TABLE IVa

PREOPERATIVE VISUAL FIELDS VS. REATTACHMENT

	AFTER FIRST OPERATION		AFTER ONE OR MORE OPERATIONS	
	NO. OF CASES	PERCENTAGE CURES	NO. OF CASES	PERCENTAGE CURES
1 Quadrant or less	91	81	90	85
2 Quadrants	145	68	150	73
3 or 4 Quadrants	79	44	83	52

$P = < 0.001$

TABLE IVb
AREA OF DETACHMENT
VS.
REATTACHMENT AFTER ONE OR MORE OPERATIONS

AREA	NO. OF CASES	PERCENTAGE CURES
1 Quadrant	89	77
2 Quadrants	194	66
3 Quadrants	55	87
Complete	54	48

TABLE IVc
SIZE OF DEFECT IN PREOPERATIVE VISUAL FIELD
VS.
REATTACHMENT

DEGREE OF DEFECT	NO. OF CASES	PERCENTAGE CURES
(1, 2 or 3 quadrants)		
20°	65	83
40°	66	74
60° +	185	65
P = Slightly > 0.05		

preoperatively (table Va). The fact that one or more breaks are detected preoperatively does not exclude the possibility of additional breaks being overlooked, and it is possible that the difference shown in table Va might be even

TABLE Va
DETECTION OF BREAKS
VS.
REATTACHMENT AFTER ONE OPERATION

BREAKS	NO. OF CASES	PERCENTAGE CURES
Not found	29	45
Found	295	65
P = <0.05		

greater if it were certain that all the breaks had been detected. However, the number of breaks found did not have any significant relation to the possibility of cure. It is interesting that the type of break had no significant effect on the prognosis (table Vb), although the size of a small number of disinsertions was of suggestive importance.

TABLE Vb
TYPE OF BREAK
VS.
REATTACHMENT

TYPE	NO. OF CASES	PERCENTAGE CURES
Round	158	71
Horseshoe	140	75
Disinsertion < 1½ hr.	31	81
" 1½ hr. → < 1 Quad.	15	67
" 1 Quad. or more	7	43
P = 0.1		

Reapposition of retina. It has been pointed out that cures are greatly dependent upon the ability of the retina to fall back into close apposition with the points of coagulation. This favorable prognostic sign has been substantiated by the results in our series. It was found that the presence of immovable retinal folds, which usually have a sharp ridge radiating out from the posterior pole, yielded a poor operative prognosis (table VIa). The degree of flattening

TABLE VIa
FIXED AND MOVABLE RETINAL FOLDS
VS.
REATTACHMENT

TYPE OF FOLDS	NO. OF CASES	PERCENTAGE CURES
Fixed	24	38
Movable	64	84
P = < 0.001		

of the retina on bed rest and the use of pinhole glasses or binocular bandaging also gave a significant clue as to the chances of reattachment after operation (table VIb). Also at the time of opera-

TABLE VIb
FLATTENING OF RETINA ON BED REST
VS.
REATTACHMENT AFTER OPERATION

AMOUNT OF FLATTENING	NO. OF CASES	PERCENTAGE CURES
None or slight	179	64
Moderate to marked	114	85
P = < 0.001		

tion after the subretinal fluid had been evacuated, the degree to which the retina fell back into normal position was related to the eventual cure (table VIc).

TABLE VIc
POSITION OF RETINA AFTER WITHDRAWAL OF SUBRETINAL FLUID ON FIRST OPERATION VS. REATTACHMENT

POSITION OF RETINA	NO. OF CASES	PERCENTAGE CURES
Relatively flat	85	78
Moderately elevated	45	62
Markedly elevated	13	46
P = 0.03		

If the retina fails to flatten out after the evacuation of subretinal fluid, the injection of saline or air into the vitreous has been recommended, but in this series, these procedures were done too infrequently to arrive at any definite conclusion about their effectiveness.

Other preoperative factors. The following additional preoperative factors were studied in relation to the result of operation and were found to be without significant effect: age varying between 10 and 80 years; race and sex; duration of the detachment as long as five years prior to operation; degree of trauma to the eye as a predisposing factor; the location of the detachment; the location of the breaks; the condition of the retina with the possible exception of diffuse shrinkage and loss of shagreen; the condition of the vitreous including fluid vitreous, strands in the vitreous, or detachment of the vitreous; intraocular tension; or positive aqueous ray.

Influence of Operative Technic On Reattachment

In the study of operative technic in relation to the percentage of cures, it was found that certain procedures were more important than possible variations in the skill of the operator. The results of operation were analyzed in an attempt to discover those technics which proved most successful.

Localization of retinal breaks. In general, several technics as outlined by Dr. Pischel and Dr. Schepens were used for the detection and localization of retinal breaks, both preoperatively and at the time of operation. A correlation of these methods, not necessarily used alone, with the results of operation is given in table VII. Worse results were

TABLE VII
METHOD OF LOCALIZATION OF BREAKS VS. REATTACHMENT AFTER ONE OR MORE OPERATIONS

METHOD	NO. OF CASES	PERCENTAGE CURES
No breaks localized	28	46
Stine	54	46
Sketch	131	73
Transillumination with ophthalmoscope	171	83
Surface coagulation	104	78
Pin	45	62
Frequent examinations during operation	74	89

obtained when no break could be localized or in those cases where the Stine method was employed. This does not imply that the Stine method is not a valuable adjunct in the localization of breaks; e.g., in determining the position of the initial surface coagulation or localizing pin, or especially if the break can no longer be seen during operation. However, the Stine method alone may not provide sufficiently accurate localization to insure sealing off the retinal break.

Diathermy. Ordinary diathermy operations were performed, except for 38 scleral resections and 3 cases of chemical cauterization (2 of which became reattached). No difference in results was apparent after using surface coagulation, 0.5 mm. partial penetrating electrodes, 1.0 mm. electrodes, pins, or a combination of these methods. The area treated by one or more of these methods at one operation showed a significant relation to the percentage of cures (table VIII). With increasing area of barrage the percentage of cures progressively diminished. Blind barrages

TABLE VIII
AREA TREATED BY DIATHERMY VS. REATTACHMENT

AREA TREATED	NO. OF CASES	PERCENTAGE CURES
1. Only break	45	80
2. Break and barrage	122	65
3. Only barrage	39	49
1 + 2 + 3 P = <	0.01	
1 + 2 P =	0.02	

were undoubtedly employed where a break had not been detected preoperatively.

It has been pointed out previously that the amount of coagulation by diathermy is difficult to measure because of variability in the machines and moistness of the operative field. Some estimation can be made by ophthalmoscopic examination of the fluffy white spot which appears in the area of coagulation. However, visibility of this white spot depends not only on the intensity of the scleral, choroidal and possibly retinal coagulation, but also the degree of retinal elevation in this area. In this series, the prognostic import of these white spots was inconclusive (table IX), although the percentage of eyes which eventually obtained 20/40 vision or better was greatest in the group where coagulation points in the retina were minimal. The intensity of retinal coagulation is not related to the development of certain postoperative complications (table X). These data should not imply

TABLE IX
INTENSITY OF RETINAL COAGULATION VS. REATTACHMENT

INTENSITY OF COAGULATION	NO. OF CASES	PERCENTAGE CURES
Not seen or mild	102	75
Moderate or marked	173	64
P = Slightly > 0.05		

that it is undesirable to produce sufficient coagulation to insure sealing of the retinal break, but rather that ophthalmoscopic evidence of scleral coagulation or diathermy punctures is unnecessary to obtain a cure.

Evacuation of subretinal fluid. The evacuation of subretinal fluid has long been considered a prerequisite for successful detachment surgery. In the present series (table XI), there was only a suggestive difference in the percentage of cures obtained after the evacuation of large amounts of subretinal fluid. It should be borne in mind that those cases where little or no subretinal fluid was obtained probably included those in which the retina flattened out preoperatively to a great extent, and it has been shown previously that this offers a good prognosis. In 7 cases where the eye became very soft or partially collapsed following the evacuation of subretinal fluid, the percentage of cures was not reduced.

In cases where subretinal fluid is not obtained after diathermy punctures have been made, the question arises as

TABLE X
INTENSITY OF RETINAL COAGULATION VS. POSTOPERATIVE COMPLICATIONS

INTENSITY OF COAGULATION	NO. OF CASES	PERCENTAGE OF POSTOPERATIVE COMPLICATIONS						
		NONE	IRITIS	CLOUDY VITREOUS	VITREOUS HEMORRHAGE	NEW TEARS	EXTENSION OF DETACHMENT	NEW DETACHMENT
Not seen or mild	28	25	7	11	4	7	29	7
Moderate to marked	154	45	0	10	4	7	25	7

to the advisability of attempting to remove subretinal fluid by other measures. In 8 cases where suction was applied over the diathermy holes by means of a medicine dropper, 6 resulted in reattachment (table XI). Should this simple maneuver fail, the operator often considers more heroic measures such as a sclerotomy or sclerectomy. A comparison of 77 cases in which a sclerotomy was performed resulted in only 54 per cent cures, in significant contrast with 71 per cent cures of 318 cases for the entire group (table XI). Only one of 7 cases in which a sclerectomy was performed resulted in a cure. These results after sclerotomy and sclerectomy are no better than those where the retina remains elevated after the withdrawal of subretinal fluid (table VIe), and it is therefore doubtful whether these two procedures are ever indicated.

Others have mentioned the danger of producing new retinal holes by perforating needles, either when the retina is relatively flat or if the pins or needles are inserted after the evacuation of subretinal fluid. The data in this series are not conclusive because of the small number of cases in which the possibility of such retinal puncture has existed (table XII). However, it is interesting that, of 9 cases in which new holes were found by ophthalmoscopic examination at the time of operation to be formed by the diathermy pins, 7 cases or 78 per cent resulted in cures. It seems likely that such holes secondary to the diathermy needles are in most cases sealed off by the surrounding chorioretinal reaction.

TABLE XI

EVACUATION OF SUBRETINAL FLUID VS. REATTACHMENT

METHOD AND AMOUNT OF SUBRETINAL FLUID OBTAINED	NO. OF CASES	PERCENTAGE CURES	
After Withdrawal of Diathermy Pins			
1. Little or no fluid obtained	78	62	71
2. Moderate to large amount of fluid obtained	240	73	
3. Eye very soft	7	72	
4. Fluid obtained by suction over diathermy holes	8	75	
5. Sclerotomy	77	54	
6. Sclerectomy	7	14	

1 vs. 2 P = 0.08
1 + 2 vs. 5 P = 0.007

TABLE XII

PERFORATING DIATHERMY PUNCTURES VS. REATTACHMENT

PROCEDURE	NO. OF CASES	PERCENTAGE CURES
Surface diathermy only	12	58
Punctures before evacuation of subretinal fluid	306	69
Punctures after evacuation of subretinal fluid	8	50
New holes formed by diathermy pins	9	78

Scleral resection. Scleral resection was performed on 22 aphakic and 7 phakic patients either as a primary operation (17 cases) or as a secondary operation after diathermy procedures had failed (12 cases). In general, a resection of 3 mm. of sclera was performed in two quadrants at one operation, and the procedure was repeated in 9 cases. Of these various combinations of operations in these difficult cases, 18 per cent cures were obtained (table XIII).

Postoperative Complications

An attempt to relate certain postoperative complications to predisposing ocular characteristics or operative technics was in general not illuminating. An exception to this was the presence of peripheral cystic degeneration in which 38 per cent of 56 cases developed postoperative extension of the detachment,

TABLE XIII

RESULTS OF SCLERAL RESECTIONS

OPERATIONS	NO. OF CASES	PERCENTAGE CURES
One scleral resection	12	25
Two scleral resections	4	25
One scleral resection followed later by diathermy operation	1	100
One diathermy operation followed later by scleral resection	6	33
One diathermy operation followed later by 2 scleral resections	5	20
One diathermy + one scleral resection + one diathermy	1	0
SUMMARY OF RESULTS OF ALL SCLERAL RESECTIONS	38	18

TABLE XIV

POSTOPERATIVE COMPLICATIONS VS. FINAL REATTACHMENT WITH OR WITHOUT REOPERATION

COMPLICATION	NO. OF CASES	PERCENTAGE CURES
All cases with and without complications	373	68
No complications noted	61	79
Iritis	2	50
Cloudy vitreous	46	56
Vitreous hemorrhage	11	54
Massive exudation	3	0
Reaction not around break	6	33
New breaks formed	20	35
Extension of detachment	62	27
New detachment	11	18
Poor cooperation	3	0

compared with 21 per cent of 107 cases in which no peripheral cystic degeneration was detected. However, several of the postoperative complications themselves appeared to have a deleterious effect on the eventual outcome (table XIV).

Comparison of aphakic and phakic detachments. It has been pointed out previously that the results of detachment operations are less successful in aphakic than in phakic eyes. A comparison of prognostically important factors in these two groups of cases is given in table XV. In general, the size of the detachments in aphakics is larger, breaks are found less frequently, detachments do not settle down as readily on bed rest and disturbances of vitreous are more common. It has been shown elsewhere that rupture of the hyaloid membrane and loss of vitreous at the time of cataract extraction predisposes to the development of retinal detachments. However, in this series for both phakic and aphakic eyes, we were unable to demonstrate any significant correlation between the condition of the vitreous preoperatively (e.g., vitreous in the wound or anterior chamber, formed versus fluid vitreous, presence of vitreous strands, detachment of the vitreous, or adhesions of the vitreous to the retinal break) and the operative result. However, it may well be that the rather gross

TABLE XV

COMPARISON OF PROGNOSTICALLY IMPORTANT CHARACTERISTICS OF DETACHMENTS IN APHAKICS AND PHAKICS

	TOTAL DETACHMENTS	NORMAL VITREOUS	VITREOUS STRANDS	HYALOID RUPTURED	NO BREAKS FOUND	FLATTENING ON BED REST	
						NONE	MARKED
Aphakic	27	14	63	43	18	11	7
Phakic	12	33	38	0	6	2	19
* P	0.01	0.003	0.003		0.05	0.04	

* P = statistical probability of error of difference between the 2 groups.
Maximum number of cases used: aphakics = 64; phakics = 312.
Figures above given in percentage of each group with the characteristics.

changes in vitreous structure noted here do not give an adequate picture of the effect of vitreous on the development or cure of retinal detachment, or that secondary changes in the vitreous may occur postoperatively, resulting in operative failure or recurrence.

Secondary operations. The likelihood of success after reoperation for detachment is given in table XVI. Although

TABLE XVI
REATTACHMENT AFTER PRIMARY AND SECONDARY OPERATIONS

NO. OF OPERATIONS	NO. OF CASES	PERCENTAGE CURES
After primary	368	62
After second	79	41
After third	13	31
After fourth	2	0
After fifth	1	0

failure after the first operation reduces the chance of cure on reoperation, the prognosis is not by any means hopeless for at least three operations.

VISUAL RESULTS

The second major criterion of successful surgery is good central visual acuity. A comparison of the preoperative and postoperative vision in all cases with adequate data is given in table XVIIa. In summary, 44 per cent of the cases showed improvement of vision, 18 per cent had worse vision, and 38 per cent had no change in vision after operation. As would be expected, the major cause of poor vision was a detachment remaining or recurring after operation, and very few cases showed a reduction of vision following successful reattachment of the retina (table XVIIb). It is rather striking that almost a third of all cases with vision of 20/200 to as low as light perception preoperatively obtained a final vision of 20/40 or better following successful reattachment. A few of these cases of poor preoperative vision may represent a bullous detachment overhanging an intact macula. However, it appears that detachment of the macula

TABLE XVIIa
PREOPERATIVE VISION VS. POSTOPERATIVE VISION (ALL CASES)

PREOPERATIVE VISION	NO. OF CASES	POSTOPERATIVE VISION (PERCENTAGE OF CASES)					
		IMPROVED	20/40 +	20/50 TO 20/100	20/200–	HAND MOVEMENTS	WORSE
20/40 or better	47	—	72	6	9	13	28
20/50-20/100	55	55	55	25	9	11	20
20/200 to counting fingers	183	49	21	28	28	23	23
Hand movements or worse	73	53	16	21	16	47	—

TABLE XVIIb
PREOPERATIVE VS. POSTOPERATIVE VISION IN CURED CASES

PREOPERATIVE VISION	NO. OF CASES	POSTOPERATIVE VISION (PERCENTAGE OF CASES)			
		20/40 +	20/50 to 20/100	20/200 –	HAND MOVEMENT OR LESS
20/40 or better	38	90	8	2	0
20/50 to 20/200	45	66	27	7	0
20/200 to counting fingers	120	31	42	23	4
Hand movements or less	37	32	41	22	5

TABLE XVIIIa
DURATION OF DETACHMENT
VS.
VISUAL RESULTS

DURATION	NO. OF CASES		POSTOPERATIVE VISION (PERCENTAGE OF CASES)					
			20/40 +		20/50 to 20/100		20/200−	HAND MOVEMENTS OR WORSE
1-13 days	94		33		24		17	25
14-20 days	47		45		19		19	17
21-28 days	28	293	43	36	15	22	15 18	28 24
1-2 months	89		34		23		18	26
3-5 months	35		37		21		23	20
6-12 months	19		0		47		37	16
1 year and over	17	36	0	0	18	33	24 31	58 36

does not necessarily indicate a poor visual prognosis. The question arises: how long must the macula be detached before loss of function occurs? Such data are difficult to obtain because the history of the onset of symptoms does not necessarily indicate the date of onset of the macular detachment. However, correlation of the duration of the detachment with final visual results (table XVIIIa) indicates that the ultimate visual prognosis for 20/40 or better vision was not affected by the history of a detachment less than six months old, but that none of 36 cases with a history of detachment dating back more than six months obtained 20/40 or better vision.

An analysis of these two groups of cases showed that, although the preoperative vision of more recent detachments was essentially the same as older detachments, the incidence of macular detachments and total detachments was slightly but significantly higher in detachments over six months old. This may well be a partial explanation of the poorer visual results in older detachments. It is possible that the macula can be detached for several weeks or perhaps a few months without complete loss of function, but none of the 14 cases giving a history of symptoms over six months and cured by operation regained macular vision (table XVIIIb).

TABLE XVIIIb
DURATION OF DETACHMENT WITH
MACULAR INVOLVEMENT CURED BY OPERATION
VS.
POSTOPERATIVE VISION

DURATION	NO. OF CASES	POSTOPERATIVE VISION (NO. OF CASES)			
		20/40 +	20/50 to 20/100	20/200−	HAND MOVEMENTS OR LESS
1-13 days	13	3	8	2	0
14-20 days	4	2	2	0	0
21-28 days	2	0	1	1	0
1-2 months	14	4	8	2	0
3-5 months	6	3	2	1	0
6-12 months	9	0	7	2	0
1-2 years	2	0	1	1	0
4 years and over	3	0	0	1	2

SUMMARY

A survey of 375 cases operated on for primary retinal detachment by members of this symposium has emphasized several of the points made in previous discussions. Those factors which adversely affect the prognosis for reattachment of the retina include (1) myopia; (2) peripheral cystic degeneration; (3) the size of the detachment, although 48 per cent of total detachments were cured; (4) failure to detect and localize retinal breaks; (5) failure of the retina to fall back in apposition to the choroid; (6) blind and possibly excessive barrage with diathermy current; and (7) sclerotomies and sclerectomies for the evacuation of subretinal fluid. The relatively poorer prognosis in aphakics compared to phakic eyes may be partially explained by their larger detachments, less frequent localization of the breaks, and less flattening out on bed rest.

The results of 38 scleral resections performed largely on aphakic eyes or after diathermy had failed showed 18 per cent cures.

Visual improvement is often dramatic after surgical reattachment of the retina, even though the macula has been previously detached.

Symposium: Retinal Detachment

DISCUSSION

DR. SCHEPENS: The first question is, "Is incidence of superior tears greater in the area of the superior oblique than elsewhere? What is your opinion of effect of superior oblique in production of these tears?"

I think it is true enough that many tears do occur in the upper temporal quadrant in the vicinity of the insertion of the superior oblique. There is no evidence, clinical or otherwise, that this insertion has anything to do with it. The only evidence we have is that equatorial degeneration seems to occur particularly in that area.

The second question is: "Do you measure with a divider or with a curved ruler how far from the limbus are (1) the anterior border of the ciliary body; (2) the posterior border of the ciliary body; (3) the ora serrata?

I believe that the distances mentioned are so small that it makes little difference whether one uses a curved ruler or a divider. The distance between the limbus and the anterior border of the ciliary body is roughly about one millimeter. The distance between the limbus and the posterior border of the ciliary body is roughly about four millimeters. The distance between the limbus and the ora serrata is about seven millimeters.

DR. PISCHEL: "Have you observed a retinal tear in a detachment associated with a tumor?" This is very rare but it does occur. There are on record cases observed by more than one man in which there was a tear or a retinal break in which a choroidal tumor was present. But I wish to emphasize that it is very rare.

The next question I think was answered by Dr. Kronfeld. "How soon should a second operation be performed if the first is immediately seen to be unsuccessful?" I can only urge, as Dr. Kronfeld did, that the operator have courage to admit that it was a failure and reoperate early; for example, within 12 or 14 days.

"In locating the hole why not have your assistant mark with a Kronfeld needle and save a step?" As I understand that question, it means that instead of marking with gentian violet or some dye, one might mark it with diathermy. That is a perfectly good procedure. We have found it a little awkward as the electrode got in the way of good observation, and the mark with the dye is satisfactory in most cases. It does bring up the question that the mark with the dye may not last indefinitely unless you get a good mark.

DR. KRONFELD: I have a very fundamental question: "Why does a separated retina not function and, when in place after surgery, see again?" This pertains to the formation and regeneration of photosensitive substances. The inner layers of the retina may be expected to function quite well even in the state of separation. The outer layers, however, depend upon pigment epithelium and choroid for their supply of nutritive and photosensitive substances.

This is well illustrated by those cases in which we have brought about a partial cure. There is a patient of ours here in Chicago who is a well-known cardiologist. In his only seeing eye the lower half of the retina is markedly elevated, whereas the upper half is fairly close to but not exactly in contact with the choroid. As long as this gentleman does not expose himself to very bright lights he does quite well. But if he goes for a stroll on Michigan Boulevard at this time of day, he is blind for the rest of the afternoon. If he keeps away from bright lights, the rate of regeneration and penetration of the photosensitive substances into the slightly detached retina can keep up with their breakdown under the influence of light.

I have a second question here, "Should scleral shortening ever be done as the first operation in retinal detachment?" I believe there are such situations. They are not common, but I do believe that one occasionally sees a detachment of the lower half of such long standing that the retina obviously is too short for the area which it has to cover. Here scleral resection is indicated as the primary operation, combined with electrocoagulation of tears located posterior to the area of the sclerectomy.

I have a third question: "Does vitreous hemorrhage causing black reflection with no fundus details at conclusion of operation commonly impair the visual result? How soon may it be expected to absorb? Two to three months or more than that?" Small hemorrhages, either into the retina or into the vitreous or between the layers of the eyeball wall are not too infrequent during and after operation. It has been my experience that hemorrhages concealing the details of the eyeground over one quadrant or less are not of any great significance; most of those clear up and the patients recovers with a good visual result if all other factors are favorable. The very massive hemorrhages occurring during the operation are very serious events and, in my experience, always related to some misfortune on the part of the operator, misfortune in the sense that he interfered either with a vortex vein itself or with its ampulla. A needle electrode may break into an ampulla and make an opening from there into the subretinal space. Under those conditions a very massive hemorrhage is very likely to occur; such eyes, I believe, have no future. That is why I do everything I can to keep away from the vortex veins. After extensive coagulations of the sclera and choroid a vortex vein may gradually become obliterated as the result of the involution of a large section of choroid. This gradual process of closure is apparently not injurious to the eye, in contradistinction to the disastrous effect of sudden closure of a vortex vein by diathermy.

NEW CONCEPTS IN THE USE OF ACTH IN OPHTHALMOLOGY AND OTOLARYNGOLOGY

GROSVENOR W. BISSELL, M.D.
CHICAGO, ILL.
BY INVITATION

ALTHOUGH the importance of the adrenal glands was known through the work of Addison and Brown-Sequard in the mid-nineteenth century, the close relation between the pituitary and other target organs, especially the adrenals, was not appreciated until approximately 25 years ago. In 1924 Evans observed adrenal hypertrophy following injection of anterior pituitary extracts in rats. Two years later Smith observed atrophy of the adrenals following pituitectomy.

Gradually, through the efforts of many workers, it became appreciated that a number of hormones such as somatotropin, thyrotropin and the gonadotropins were present in the pituitary, and in 1933, Collip was able to delineate adrenocorticotropin or ACTH as a pituitary hormone. However, due to inadequate assay technics ACTH remained as a laboratory curiosity for a number of years to follow. Actually Armour Laboratories began work on ACTH in collaboration with Collip in 1937, but progress was slow until the assay technics of Sayers made the isolation efforts of the biochemists easily followed.

In 1943 Browne and his group in Montreal demonstrated a stimulation of certain adrenal cortical functions by exogenous ACTH. In 1947, the Thorne and Albright groups reported a stimulation of all known cortical functions by the parenteral administration of ACTH.

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Presented at the Fifty-Sixth Annual Session of the American Academy of Ophthalmology and Otolaryngology, Oct. 14-19, 1951, Chicago, Ill.

The physiological effects of ACTH may be summarized as follows:

Under stimulation of ACTH the adrenal cortex increases the production of all its hormones, which can be grouped into three large categories:

1. The androgenic steroids as reflected by the increased excretion of 17-ketosteroids in the urine.
2. The salt and water steroids as reflected by sodium retention and potassium excretion.
3. The sugar or Compound E and Compound F hormones as reflected by increased corticoid excretion, increased excretion of uric acid and a lysis of the circulating eosinophils, the latter of which makes possible a clinical estimation of the degree of adrenal stimulation.

It is the stimulation of this third group of hormones by ACTH which accounts for similar clinical effects of ACTH and cortisone, which is Compound E and hydrocortisone, which is Compound F.

This stimulation of Compound F substances by ACTH becomes of interest in relation to inflammatory diseases and the diseases of hypersensitivity.

The allergic manifestations of hypersensitivity are virtually abolished by the stimulation of Compound F substances by ACTH. Local swelling, itching, and blister formation are diminished or obliterated. An antigen such as tuberculin may be given to a patient on ACTH without any hypersensitivity response. These activities obviously make this a powerful agent in combating the hyper-

sensitivity states, which include allergies, contact dermatitis, Rhus toxicodendron dermatitis, etc.

In addition there is a marked modification of the inflammatory reaction. Perivascular exudate is diminished. Pus formation is minimal despite marked leukocytosis. There is inhibition of fibroblastic proliferation. However, through the inhibition of the hyaluronidase system, local spread is decreased.

It is obvious from this that the basic manifestations of ACTH stimulation allow formidable clinical application. In otolaryngology, such conditions as allergic rhinitis, allergic sinusitis, hay fever, asthma, and status asthmaticus may be controlled easily.

By the same token, numerous ocular conditions due to inflammatory disease may be cured or modified. In general, the more acute oculopathies are more amenable to permanent cure, whereas more chronic disease may only be modified and demand prolonged therapy.

It should be remembered also that because of the masking of the usually expected inflammatory reactions by ACTH, underlying infection may be masked, and, therefore, adjunctive antibiotic therapy should be given, especially to patients receiving ACTH for prolonged periods.

In the actual application of the material clinically it may be well to state that there is no set rule of thumb, and patient response is considerably variable.

ACTH may be given in a dose of 25 to 50 mg. intramuscularly every six hours around the clock; or 20 mg. in 500 cc. of 5 per cent glucose in distilled water intravenously over an eight-hour period. A long-acting preparation in gelatin often necessitating only one injection daily is now available.

Cortisone may be given intramuscularly or by mouth. In ocular conditions it may also be administered topically or by subconjunctival injection. ACTH has no topical application.

In conclusion, it should be re-emphasized that the clinical usefulness of ACTH and cortisone is due to their effects in altering tissue reaction to hypersensitivity and inflammation. It should also be remembered that once fibroblastic proliferation, collagen formation, or degeneration has occurred, regeneration of normal tissue should not be expected.

Doctor Dan Gordon, who is tied up with a course at this moment, has asked me to present the following case for him.

The first picture shows the patient before institution of treatment with 100 units of ACTH in Gel daily. At this time the vision was bare light perception.

The second picture is of the same patient after 8 days of treatment. Tremendous improvement in vision and marked improvement in the appearance of the optic nerve has occurred.

In using either ACTH or cortisone, it is important to bear in mind these two very important facts. First, that if one of these hormones fails, the other may succeed. The common error is that in having failed with one, the physician assumes that the other also fails, and will not use it. Secondly, one must have a definite plan of attack. If an acute disease process does not respond within 48 or 72 hours after treatment is instituted, we immediately either increase the dose of the hormone being used or switch to the alternate hormone, or both in succession, if need be.

In our experience, intravenous ACTH is the most potent of the hormonal weapons now available.

DISCUSSION

AUBREY G. RAWLINS, M.D., San Francisco, Calif.: My discussion of this paper is to be limited to the eosinophil test. This is commonly referred to as the "Thorn test."

Almost any stress will produce, through the hypothalamic-pituitary-adrenal cortex mechanism, a lowering of the number of circulating eosinophils in the patient. ACTH and cortisone given to a normal person likewise produce an eosinopenia. Intravenous or subcutaneous injections of adrenalin also produce, in the normal person, a similar result. Adrenalin stimulates the pituitary to liberate ACTH, which in turn stimulates the adrenal cortex to produce cortisone (probably Compound F). In other words, anything that stimulates the normal adrenal cortex to produce Compound F or cortisone lowers the number of circulating eosinophils.

The eosinophil response is the most useful test for determining the activity of the pituitary-adrenal stress mechanism in man. In cases of very acute allergy, where the circulation is being flooded with eosinophils, the test may not be quite so accurate.

The ACTH test is as follows: After an original eosinophil count, give 25 mg. of ACTH intramuscularly. Four hours later another eosinophil count should show a drop of at least 50 per cent in normal individuals.

The adrenalin test is as follows: After an original eosinophil count, give 0.2 cc. of 1:1000 adrenalin intravenously in 200 cc. of saline over a one-hour period. An eosinophil count should be repeated in four hours from the beginning of the infusion.

Instead of this method, 0.3 cc. of 1:1000 adrenalin may be given subcutaneously and an eosinophil count repeated in four hours. There should be a decrease of at least 50 per cent in circulating eosinophils if the pituitary-adrenal mechanism is sufficient. In Addison's disease, these tests usually show no decrease, or a very slight decrease in circulating eosinophils. If the ACTH test shows a normal response and the adrenalin test does not, the pituitary is not functioning properly.

In treating patients with cortisone and ACTH, the eosinophil drop is usually a good indication of a proper response to treatment, and usually the symptoms subside accordingly.

If ACTH does not reduce the eosinophils normally and symptoms do not subside, but cortisone is used with success, then the adrenal cortex is hypoactive. That emphasizes the point that Dr. Bissell made, if one does not work, you should try the other.

An example of this in an asthmatic male, aged 65 is as follows: His pretreatment eosinophil count was 345 per cubic millimeter. After receiving 25 mg. of ACTH intramuscularly every six hours for forty-eight hours, he showed no improvement in symptoms. His eosinophil count was still 350 per cubic millimeter. The dose was increased to 40 mg. of ACTH every six hours. After twenty-four hours there was a slight improvement, and the eosinophil count had dropped to only 220 per cubic millimeter. ACTH was discontinued, and cortisone was given in doses of 25 mg. every six hours for twelve hours. At this time the patient's wheezing had subsided and the eosinophil count had dropped to 50 per cubic millimeter.

The following is an example of a normal eosinophil response to moderate stress. The patient was a male, aged 45. His prestress count was 189 per cubic millimeter immediately prior to a Caldwell-Luc antrum operation of one-hour's duration under intravenous sodium pentothal anesthesia. Four hours later it was 55 per cubic millimeter. Eight hours later there were 30 eosinophils per cubic millimeter. Three days later the count was 230 per cubic millimeter. This shows a normal response.

The ACTH test may be used to determine whether or not a patient can well tolerate a major surgical procedure or whether a patient in emergency straits might need cortisone. Give 25 mg. of ACTH intramuscularly, and if the eosinophils haven't dropped 50 per cent in four hours, the patient probably will not tolerate much stress.

HENRY L. WILLIAMS, M.D., Rochester, Minn.: I enjoyed Dr. Bissell's presentation of the physiology of ACTH and cortisone very much and he presented many illuminating points. I should, however, like to enter a mild objection to some of the statements which he made in his paper, particularly in regard to the suggestion as to the indiscriminate use of cortisone, particularly by otolaryngologists. He did stress, of course, some of the contraindications to the use of cortisone and ACTH therapy. However, one which has appealed to us as of some importance is the fact that in patients who have received cortisone therapy, such therapy produces a hypoactivity of the adrenal and they are not suitable patients for surgery until they have undergone a suitable recovery period of several weeks. These patients have to be handled as though they had Addison's disease if emergency surgery is necessary.

I also think that it is somewhat misleading to suggest that such diseases as hay fever, vasomotor rhinitis, and some of the other minor conditions of ear, nose, and throat practice are easily relieved by the use of ACTH or cortisone. We have been somewhat disappointed in the use of these substances in these particular conditions. In the summers of 1949 and 1950 we treated a series of cases with vasomotor rhinitis and asthma with very marked improvement in their symptoms. This last summer the same group of patients were treated with both cortisone and ACTH with a much less dramatic improvement in their symptomatic relief.

The possibility of developing resistance to cortisone was suggested by McNally in a paper which he wrote in conjunction with Bram Rose and others. In treating vasomotor rhinitis with polyposis, they found that a patient might be relieved by a first treatment with ACTH or cortisone; with the second treatment the response would be less dramatic, and on the third treatment the patient would show no response whatever to medication with cortisone or with ACTH. Some individuals have apparent initial resistance to these substances and do not respond to them at all well. This is illustrated by a small group of five patients whom we have been treating with cortisone for vasomotor rhinitis. Two of them showed no appreciable clinical response to this medication, although they received 200 mg. in twenty-four hours, in four divided doses by mouth. Two of them showed a response which we estimated to be about 50 per cent, with a slight reduction of the polyps, and only in the fifth case was there a result which might be considered clinically satisfactory.

We already have routine methods of treatment of these conditions, with results which are moderately satisfactory clinically. I hardly think that we would be justified in putting our patients to the expense of therapy with cortisone or ACTH over a two-week period to secure a somewhat improbable and very evanescent result. McNally stated that in his somewhat larger series the disappearance of polyps lasted about six weeks, after which time the condition was as before treatment. I think, therefore, that the otolaryngologist ought to reserve the use of these very potent hormones for those conditions in which it appears their use might be a life-saving matter. Those three conditions which seem to fall in this category in my experience are status asthmaticus, pemphigus and lethal granuloma of the midline facial tissues. Status asthmaticus is only rarely in charge of the otolaryngologist, and this might also be said of pemphigus, although we are a little more likely to be in charge of a case of the latter. Midline lethal granuloma appears to fall frequently into the lap of the otolaryngologist. Many commentators on the use of ACTH and cortisone have appeared to be dissatisfied with the fact that there was no unifying concept by which such apparently diverse disorders as hay fever, asthma, periarteritis nodosa, pemphigus, lethal granuloma of the midline facial tissues, or lupus erythematosus disseminatus could be treated on a logical basis. I think, however, that there is such a basis and that we can use it in exploring the possibility of the use of these substances in the disorders which we treat. All inflammatory reactions are affected by the use of cortisone and ACTH. In these conditions which I have enumerated we find localized areas of inflammation. Patients with these conditions do not have Addison's disease; they have sufficient hormones of the pituitary and adrenal cortex to carry on the normal physiologic reactions in most regions of the body. Here and there in these individuals, however, we find areas in which the physiologic reaction is abnormal and localized inflammatory reactions appear. I have presented a suggestion elsewhere that these local inflammatory reactions are due to local dysfunction of the autonomic system. While the mechanism of such localized disorders of the autonomic nervous system is unknown, it is reasonable to suppose that it involves the peripheral vascular bed as part of the autonomic system as defined by Petersen. We find in all the conditions enumerated which are favorably affected by the use of ACTH and cortisone such instances of autonomic dysfunction. I have suggested that such localized autonomic dysfunction is synonymous with allergy. Therefore, all these conditions which have responded favorably to the use of ACTH and cortisone may be classified as allergic in the broader sense of that term.

I think it would be unreasonable for the otorhinologist or other physician to begin treatment with one of these substances unless there was some reasonable evidence that the condition to be treated might be included in this classification of allergy. In lethal granuloma, in which I am particularly interested, Dr. Moore and his associates and I have been able to report what we believe is the first reported cure of this terrifically malignant and disfiguring condition. In these cases a relentless destruction of the midline facial tissue was shown by Weinberg in Baltimore to be associated with periarteritis nodosa in other areas

of the body. By the use of an initial intramuscular dose of 300 mg. of cortisone, given in twice daily doses, with the continuation of 200 mg. a day for six weeks, this heretofore fatal condition appears to be controllable.

I would reiterate, however, that I believe that our use of these substances should be restricted to those conditions in which without their use there is apt to be a fatal outcome. I disagree with the statement that we have gotten out of the era of clinical investigation in the use of these substances, and I would suggest that the average otolaryngologist might delay a little in his indiscriminate use until their effectiveness has been investigated further and until any untoward effects that might be produced have been ascertained in those institutions that have facilities to treat such conditions and to estimate properly the possible clinical result.

JOHN E. BORDLEY, M.D., Baltimore, Md. (by invitation): In discussing Dr. Bissell's report, it would be well to talk from one's own clinical experience with the administration of ACTH. The final evaluation of the excellence and the dangers of such new compounds should be made in the clinic using the ill human being as the subject. Some of the facts thus arrived at would seem relevant to this discussion.

In order to ascertain the effect of ACTH upon the tissues of the upper respiratory tract, it was decided in June 1949 at the Johns Hopkins Hospital to have all patients undergoing ACTH therapy, regardless of their disease, undergo systematic otorhinological study. There were two basic reasons for this: (1) the upper respiratory tract offers an opportunity for the direct observation of tissue changes taking place under ACTH therapy; (2) the Department of Otology was anxious to study changes that might take place under such conditions. Many changes in the upper respiratory tract were observed during these studies.

Sixty odd patients have been under observation since June 1949. Some 575 complete examinations have been made of the upper air passages. Specimens for biopsy have repeatedly been taken from the lymphoid tissue of the nasopharynx, and nasal polyps have been removed periodically throughout the hospitalization period.

Some uniform changes have been noted in all the patients under treatment. The nasal mucosa develops a dusky appearance, swelling and discharge are reduced, and clear mucus covers the mucosa. Crusts and ulceration in the nose clear up. The lymphoid tissue in the nasopharynx develops an orange-pink hue, the crypts become more apparent, and this tissue can be easily delineated from the surrounding structures. The contrast is not unlike the color changes seen in the pharyngeal lymphoid tissue in infectious mononucleosis.

Twenty-four patients suffering from respiratory allergy illustrate very well Dr. Bissell's description of changes taking place in tissues in the hypersensitivity state under ACTH stimulation. Fifteen of these patients exhibited marked polypoid changes in the nasal mucosa, and collections of polyps in each nostril. They suffered from severe nasal and postnasal discharges, and one got the picture of pale edema, on nasal examination. All 15 individuals in the presence of adequate levels of ACTH began to improve within 24 hours. There was a rapid loss of nasal edema, and decrease in discharge. Within two or three days the polyps became pinkish and began to regress, eventually either disappearing or shrinking to a very small size. The following slides illustrate the findings as seen through a nasopharyngoscope. (Slides)

Such improvement began to appear with the development of eosinopenia. Subsequent rise in the eosinophil count did not alter the rate of improvement during the course of therapy. Such improvement does not persist.

Dr. Bissell's remarks about the masking effects of ACTH on inflammatory reactions was well illustrated by the fact that 11 patients developed evidence of acute sinusitis while under therapy. Two of these developed blood stream infections. Their infections caused no pain or other warning symptoms aside from fever and some discharge. Patients are now covered by antibiotic administration at the least suggestion of fever.

Finally, the microscopic studies of the lymphoid tissue removed during treatment showed no significant uniform changes. Twenty-one nasal polyps removed from five individuals showed nothing unusual aside from some perivascular grouping of eosinophils in a few specimens.

HERMAN SEMENOV, M.D., Beverly Hills, Calif.: With your permission, I should like to show a few kodachrome slides dealing with injection of cortisone directly into nasal mucous membrances.

(Slide) This patient, as you can see, has a tremendous amount of obstruction; her nose is so completely filled with polyps and polypoid tissue that no air whatsoever can pass through it. Even the septum is involved and the polyposis measures almost an inch in thickness. She has had this condition for many years and has been treated by well-known

rhinologists, who tried almost everything.

(Slide) The nasal secretions are loaded with eosinophils, and the smears are typical of a mixed allergy and infection.

In a chronic condition such as this one, I had hoped that topical cortisone therapy might be the answer because it can be maintained over a long period of time. We tried sprays and packs for a while but it did no good. Accordingly, 5 mg. of cortisone was injected into the polypoid tissue of the nasal septum. After a few days the dose was increased to 10 mg. and then to 15 mg., etc. The moment that she received 25 mg., a violent reaction developed. Within ten seconds her face and neck became red and flushed (the way that you see it after a large dose of niacin); she complained of a sensation of substernal pressure; collapsed and fell to the floor, wheezing and almost pulseless. The same reaction was observed by Dr. J. Wall in 2 other patients, both of whom received 25 mg. of cortisone intranasally. About 40 cases received 25 mg. and 3 developed almost identical systemic reactions, which is no mere coincidence. The reactions subsided within an hour.

I continued giving the cortisone but reduced it to 5 mg. every other day. At the end of one month a little breathing space appeared, and I was able to inspect the interior of the nose and remove polyps with ease.

(Slide) Here you see a radiograph of her nose and sinuses just before cortisone therapy was started, and here is one immediately after it took effect.

(Slide) We sectioned the polyps and studied the specimens before and after cortisone was given. Except for a slight decrease in the edema, I could see no noteworthy changes in the histology.

(Slide) The histopathology, as you can see in the microphotograph, is typical of allergic polyps and consists of catarrhal desquamating epithelium, thickened hyalinized basement membrane, which I consider a collagen change, along with the striking edema of the tunica propria and disturbance of the intercellular substances.

(Slide) The swelling in the collagen tissue is what makes a polyp look like jelly, and the proliferation of new fibroblasts causes the so-called myxoid tissue.

(Slide) Here is a new-formed lymphatic follicle, which certainly is an abnormal histologic structure in this part of the nose.

In short, about the only thing accomplished by the injections was a temporary shrinkage of the membranes, which is attended with so much danger that I do not advise it.

Before concluding, I should like to ask some questions. What, if any, connection is there between the reaction mentioned above and the pharyngeal pituitary gland (accessory pituitary gland tissue) that is found in the noses of more than 95 per cent of people?

What, if any, connection is there between the pharyngeal pituitary and the involution of pharyngeal adenoids? What is status thymicolymphaticus?

DR. BISSELL: In commenting on Dr. Rawlins' remarks I am very glad that he brought out the great value of eosinphil tests in the following of the clinical efficacy of therapy with ACTH. That test is a very excellent means of knowing when you do have adequate adrenal stimulation. However, as he mentioned, in allergic states it may be of little value, and I can cite two other instances in which it may not be of value. First, you will occasionally see a patient who will respond clinically but the eosinophils will not do very much. That is a little rare but it has been observed. Second, there are desperately ill cases, such as those of acute disseminated lupus erythematosus, in which there may be few, if any, eosinophils to begin with. In these instances one is forced to go along on clinical response.

I am very grateful to Dr. Williams for his comments about the suppression of the adrenal by the use of large doses of cortisone. He is certainly quite proper and correct in saying that an individual who has been under massive cortisone therapy has adrenal suppression and should not undergo any major surgical procedure until the adrenal has had a chance to recover. Often this process may be accelerated by giving ACTH to stimulate the adrenal.

As to Dr. Williams' comments and Dr. Bordley's comments about being more conservative in their use of ACTH and cortisone in otolaryngology, it just goes to show what happens when an endocrinologist walks into a meeting of a very highly specialized group of doctors who know much more about their specialty than he does. That is why I said at the outset, as a preface to reading my formal remarks, that I was certainly not capable of discussing specific ophthalmologic and otolaryngologic entities in which ACTH or cortisone was indicated but would confine myself to a general discussion of their known physiologic and pharmacologic actions which make their clinical application so widespread.

I think I am in agreement with Dr. Williams as far as the otolaryngology goes, that perhaps most of the more conservative means of therapy would be still as effective in this particular field.

I was told that this would be a mixed group of ophthalmologists and otolaryngologists. Although the use of ACTH and cortisone perhaps is not as widely applicable in the field of otolaryngology, in the field of ophthalmology these drugs may be dramatic. I think, and this opinion is based in part on an unpublished series of some 800 cases of Dr. Dan Gordon of New York, that the patient should not be denied use of these two materials, particularly for the acute inflammatory and allergic diseases of the eye, because of fear on the part of the physician or by the fact that some phases of their use are still being investigated. That they are potent I shall not deny. That they have complications I should certainly be very foolish to deny. However, I think everyone will agree that vision should not be lost because of fear of their potency. It merely means that physicians in all medical specialties must learn how to use these materials correctly when the indication arises.

Perhaps this would be an appropriate time to emphasize that patients receiving these hormones should be given a low salt, adequate protein, moderate carbohydrate diet and should receive added potassium, say about three to five grams of potassium chloride daily.

I also should like to re-emphasize something Dr. Bordley mentioned about the inflammatory reaction because I think it is important, particularly in patients receiving ACTH or cortisone for a fairly long period of time. There is very great need to watch those patients very carefully for the masking of an inflammatory reaction due to infection. In our clinical practice we have always routinely put patients receiving long term high dosage ACTH and cortisone therapy on antibiotics.

HEREDITARY PROGRESSIVE NERVE DEAFNESS

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and
F. E. STEPHENS
BY INVITATION
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A FORM of progressive nerve deafness which appears in early adulthood occurs in some families with a moderate degree of frequency. In an attempt to determine the genetic pattern involved in such cases, two large kindreds* have been studied.[4,11]

Albrecht[1] studied ten kindreds showing nerve deafness of this type. The number of individuals with whom he worked was small and he was unable to examine all of the members of the kindreds. On the basis of his limited data, he hypothesized a dominant form of inheritance for nerve deafness. The age of onset of deafness in Albrecht's patients was not clear. Dean and Bunch,[3] Shambaugh and Shambaugh,[10] and Eagleton[5] have also contributed to the literature on hereditary nerve deafness. All presented evidence that the disease is inherited as a mendelian dominant trait.

Altmann,[2] in an analysis of the histopathology of nerve deafness, presents evidence that there are two types of pathologic change associated with this condition. The first involves primary changes in the cochlear duct and saccule. It is closely connected with deaf-mutism in man. This type of hearing defect was reported by Lindenov,[7] Macklin[8] and Hopkins[6] to be a recessive trait. The second type shows degenerative atrophy of the spiral ganglion and cochlear nerve fibers. No conclusion can be drawn as to the type of involvement until autopsy material becomes available.

Methods of Study

Utah presents a unique opportunity for the study of traits in large kindreds for several reasons. First, as in most agricultural areas, large families have been the rule. Second, in many instances families are of polygamous origin. Third, there is a great interest among Mormon people in the keeping of family records.

In Kindred I the investigation was stimulated by a common interest in the defect on the part of a geneticist, an otolaryngologist, and some affected members of the kindred. Deafness was found in Kindred II during an investigation of hereditary pyelonephritis.[9] Kindred II comprises the same population referred to in a previous publication[9] as Kindred 119 (figure 2), a numerical designation employed in our Laboratory. The method of procedure in making examinations was to have members of the kindreds gather in small groups, either at the physician's office or in the homes of the members. Each individual was questioned separately about members of his family. The information obtained in this way was checked against information from other members of the family and verified, where possible, by an examination. The examination included a personal ear, nose, and throat history and physical examination in-

From the Laboratory for the Study of Hereditary and Metabolic Disorders and the Departments of Medicine and Surgery of the University of Utah College of Medicine. This study was aided by a grant from the U. S. Public Health Service, Research Grants Division.

*A kindred includes all the descendants of one individual.

Presented at the Fifty-Sixth Annual Session of the American Academy of Ophthalmology and Otolaryngology, Oct. 14-19, 1951, Chicago, Ill.

cluding nasopharyngoscopic examination and audiogram. When this was done in a home, a quiet room in a secluded portion of the house was selected for the examinations.

In order to check the accuracy of audiograms, 5 cases were selected at random and checked again three to five months after the initial examination. The greatest deviation from the original audiograms was found to be 5 decibels, well within the limits of error. Ninety-three members of Kindred I and five individuals who had married into the families, and sixty-two members of Kindred II and two of their in-laws were studied.

Description of the Trait in Kindred I

An Englishman, born in 1788, immigrated to Utah with the early pioneers. Three hundred and nineteen of his descendants have been traced, and 72 have been found to be deaf. Of these, 10 were deaf from causes other than hereditary nerve deafness. Nineteen other members of the kindred not classified as deaf were found to have a marked high tone loss. If the numbers of deafened keep increasing, they will soon represent a public health problem.

The original progenitor had eight children, three of whom were deaf, two died in infancy and one died at 14 years of age. Nothing is known of the other two or their descendants. (See figure 1.) Of 33 individuals in the third generation, 11 were deaf. In the fourth generation, 39 out of 123 individuals were affected. In the fifth generation the ratio of affected individuals was 36 to 118. The sixth generation of 36 children has already shown 2 affected members. Many members of the fifth and sixth generation are, however, still too young for the deafness to have become apparent.

Family opinion places the age of onset of the deafness at about the age of childbearing. Since they noted that it occurs equally often in both sexes, pregnancy can not be considered a contributing factor. The deafness apparently increases with age but never becomes total.

In an effort to check on the family opinion, 93 individuals were examined. The age of onset was found to be in the early twenties. While it was occasionally monaural in the beginning, it ultimately affected both ears equally. Deafness never became total, and while severe enough to require a hearing aid in some of the older members of the kindred, it was never so severe that a hearing aid was of no help.

Tinnitus was not always present. About one-third of the individuals who developed the hearing defect stated that they had this symptom. However, some of the normal individuals in the kindred also had tinnitus.

Another defect was found among the younger individuals, particularly those in their teens. This was a high tone loss not reaching the spoken-voice range. One member of the kindred described the defect as the inability to hear violins in an orchestra.

Of the 93 individuals examined, 22 per cent showed a high tone loss; 24 per cent showed nerve deafness; 9 per cent had developed deafness from other causes, and 45 per cent had normal hearing. "Premature aging" has been used in many instances as an explanation of the mechanism of change in diseases of unknown etiology. One hesitates, therefore, to employ this term to explain the dynamics of the developing deafness in this kindred. It does, however, seem to be a possible explanation.

Ten members of the kindred have developed hearing impairment from other causes. Five of these had repeated otitis media as children and showed definite conductive hearing loss. An entirely different audiogram curve was determined

for these individuals than was obtained for those suffering from the hereditary nerve type of deafness. Three other individuals suffered from acoustic trauma. One was a miner whose job had been to detonate charges of explosives for many years. The other two returned from military service with this type of hearing impairment. These all had the V-shaped dip at 4096 double vibrations per second. The last two had post-traumatic deafness following fractured skulls where the ear drums had been ruptured and there had been bleeding from the ears.

Possible Causative Factors Other Than Hereditary

A search was made in an effort to relate the loss of hearing to other than hereditary factors. Tonsils, adenoids, allergy, malocclusion, and sinusitis were all investigated in detail as possible causes, but no association was found. A deviated septum, interestingly enough, is present in 34 per cent of the individuals who are deaf as opposed to 23 per cent in the entire group. Thirty-one per cent of those with deviated septums were also deaf. It is difficult to explain the association of deviated septums with

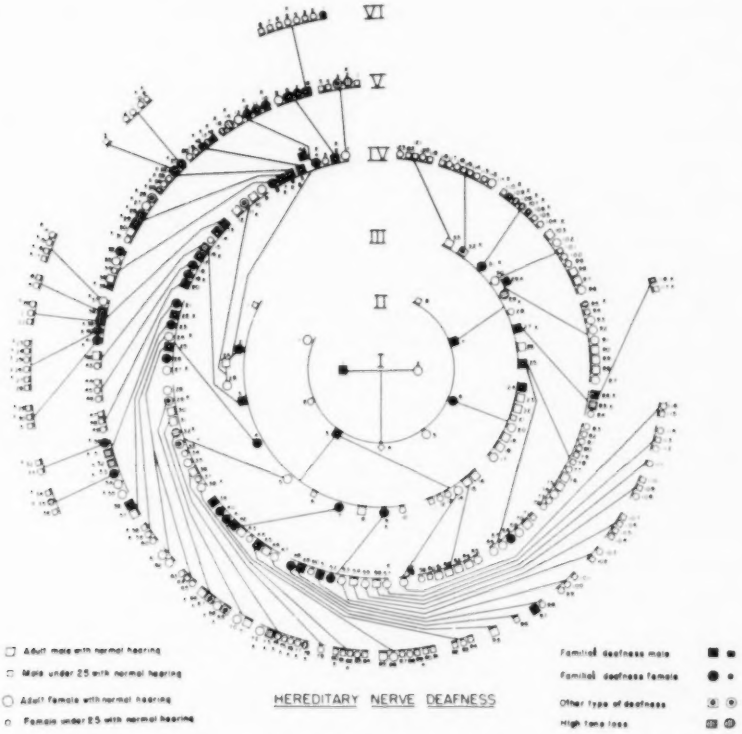


FIG. 1—Hereditary nerve deafness in a Kindred (112, Laboratory designation) referred to in this paper as Kindred I.

nerve deafness. Whether or not there is a genetic relationship is not clear. Further study of this aspect of the problem is planned.

The loss of teeth showed a striking association with nerve deafness in this kindred. There were 19 edentulous people who were examined, and of these, only one had normal hearing. These individuals were in the older age group and many had their teeth removed in an effort to prevent the progression of the deafness. Nevertheless, it is difficult to explain the striking correlation between loss of teeth and nerve deafness in so many cases.

An investigation showed no association of diabetes, nephritis, and metabolic diseases with nerve deafness. Blood types and Rh factors were determined; however, there were insufficient data to determine any relationship one way or the other between nerve deafness and these characteristics.

Inheritance of the Trait

The problem of determining the type of inheritance was complicated by several factors. First, individuals in the kindred cannot be evaluated with respect to the disease until they are in their early twenties or beyond. Secondly, some of the members of the kindred were deceased and others were not available. Finally, other types of deafness were introduced, making it necessary to diagnose each case with care. For example, an individual who was afflicted with hereditary otosclerosis married into the kindred. Until this type of deafness was recognized, considerable difficulty occurred in analyzing the data.

The data indicated that nerve deafness in this kindred is inherited as a simple mendelian dominant trait with complete penetrance. Since there was no instance of two deaf individuals marrying, the crosses were all back crosses which should give a 1:1 ratio of normal to affected offspring. In every case of hereditary nerve deafness, one of the parents was deaf.

In order to test statistically the expected 1:1 ratio, 22 families were selected in which one parent was deaf and in which there was an opportunity for testing deafness in the offspring. In all there were 122 offspring. Fifty-five of these had hereditary nerve deafness, 5 suffered from other types of deafness, 8 had high tone loss and 41 had normal hearing. If we consider all but the 55 individuals with nerve deafness as having nonhereditary forms of deafness, the ratio would be 55:67. This is a deviation of only 6 from the expected 1:1 ratio, which is not significant. If the 8 individuals having high tone loss are added to the 55 affected with nerve deafness, the ratio would be 63 affected to 59 normal, an almost perfect 1:1 ratio.

Kindred II

Kindred II presents a more complex picture of hereditary deafness. The original progenitor married two sisters. While the descendants of one have been normal, 50 of the 234 descendants of the other have had interstitial pyelonephritis and 14 have had hearing impairment. In this kindred deafness is restricted to males, though the pyelonephritis appears in both sexes. Tragically, after puberty the pyelonephritis worsens rapidly in the males, usually causing death in the thirties.

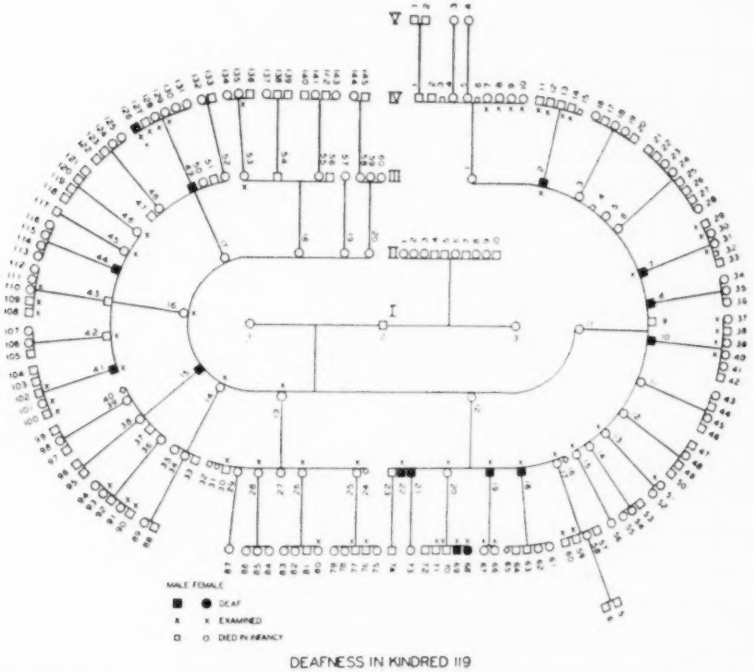
Description of the Trait

To reiterate, the original progenitor had two wives, the first of which had 10 normal children whose descendants remained free of these traits, and the second wife, a sister of the first, had 10 children, one of whom had impaired hearing and two had pyelonephritis. (See figure 2.) Of 60 individuals in the third generation from the second wife, 11 were deaf and 14 were affected with

pyelonephritis. Only one of the 145 members of Generation IV showed the hearing impairment and 34 showed pyelonephritis. Figure 2 shows three deaf members of Generation IV, but the two descendants of patient III-20 are excluded because the father had otosclerosis. The small number of individuals showing nerve deafness may be attributed to the lack of maturity of the

able to suggest, with any degree of accuracy, the age of onset. The deafness usually reaches only a moderately severe stage. However, the deaths from pyelonephritis occur in the third and fourth decade as a rule, and consequently there is little time for severe deafness to develop.

Tinnitus was not found in association with deafness. Only one member of the



DEAFNESS IN KINDRED 119

FIG. 2—Hereditary nerve deafness in a Kindred (119, Laboratory designation) referred to in this paper as Kindred II.

members of this generation or to the lethal nature of the gene. So far no hearing impairment has appeared in the six individuals who comprise Generation V.

The relatively small number of individuals in the kindred with hearing impairments has made the analysis of the trait difficult. The family has been unkindred, a woman 64 years of age with the added complication of parkinsonism and a history of mastoiditis, complained of ringing in the ears. The three others who mentioned the presence of tinnitus all belonged to a family descendant from II-12 and her husband, who had otosclerosis.

In Kindreds I and II a high tone loss

appeared in affected individuals. In the latter, however, there seemed to be two classes of audiograms. The first, similar to Kindred I, indicated a gradual loss of perception of the higher tones. In the second class a V-shaped dip at 4096 double vibrations per second which spread in both directions was shown on the audiograms. Eight individuals belonged to Class I and three belonged to Class II. The significance of these variations will have to be determined by future studies. In the latter three individuals no history of exposure to noise or explosion other than that occurring on occasional hunting trips could be elicited.

Of the 62 members of the kindred examined, 11 (or 18 per cent) showed a high tone loss, 4 (or 6 per cent) showed hereditary nerve deafness and 3 (or 5 per cent) showed hearing impairment of another type. These were the two otosclerotic children and grandchild of a man with this disease who married into the kindred.

No correlation was found with presence or absence of tonsils, adenoids, allergy, malocclusion, deviated septum, sinusitis, tinnitus or teeth. The study of the trait in Kindred I suggested that premature aging may have been the cause of deafness. So far, studies in Kindred II would indicate a different mechanism for the deafness. Further studies are necessary to determine the possible cause.

Inheritance

The data, considered by themselves, suggest that deafness in Kindred II is inherited as a sex-linked recessive trait. Since the gene for pyelonephritis is inherited as an incompletely sex-linked dominant, it is located on the X as well as the Y chromosome. From the data now available it is not clear whether the deafness is due to a separate gene or whether it is a secondary effect of the gene causing the nephritis. As the younger generations mature, further data will be collected in an attempt to clarify this point.

SUMMARY

Two kindreds showing nerve deafness have been reported. In Kindred I a definite pattern of inheritance was indicated. The trait was inherited as an autosomal dominant with complete penetrance. In Kindred II nerve deafness occurred only in males and was due to a sex-linked recessive gene or was a secondary effect of pyelonephritis. Which of these possibilities is correct cannot be stated with any degree of certainty until the younger members of the kindred become old enough to develop the trait. It is clear, however, that there is more than one mechanism producing "hereditary" progressive nerve deafness of later life.

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DISCUSSION

VICTOR GOODHILL, M.D., Los Angeles, Calif.: It is a distinct privilege to listen to this unique presentation by Dr. Dolowitz. It is probably the first time in history that a complete study of six generations of deafness has been presented.

There are few localities in the world where it is possible to carry on genetic studies with as much accuracy as in the Mormon community of Utah. In this region, genealogical records have been scruplously maintained, and geographic and sociologic unity affords an opportunity for study of several genetic diseases. The review of two kindreds with marked hereditary progressive nerve deafness is indeed a laudable and valuable addition to the otologic literature.

It is of great interest to speculate as to the numerous varieties of genetic nerve deafness that probably occur. Two basic types of genetic nerve deafness have been recognized. The first is the type due to some distinct partial or complete cochlear or auditory neural pathway aplasia which is present at birth and which results in profound infantile nerve deafness at birth. The second is the so-called *heredodegenerative* group in which we see an infantile type of nerve deafness which may come on early in the first or second year of life and an adult type which comes on in the second and third decades of life. Dr. Dolowitz' series is confined to the latter group. In no member of either kindred was there any apparent history of infantile profound nerve deafness.

The evidence produced by the study shows rather conclusively that the etiologic factor in these kindreds was a genetic trait. There must be an apparent specificity genetically for this type of cochlear degeneration, characterized by high tone perceptive impairment.

There have been relatively few attempts at genetic study of this type of neural deafness. Most prior attempts have been limited to the infantile group. Investigators at the Clarke School, including Hopkins and Macklin, have published valuable statistical studies of the genetic aspects in young children. Dr. Dolowitz, however, deals with an entirely different specific type of nerve deafness in which the onset is in the second and third decades of life and in none of which is the deafness profound to the point of complete disability. In the most severe cases these individuals could be rehabilitated with hearing aids.

We will look to Dr. Dolowitz for future pathologic reports when it will be possible for him to obtain temporal bones and brains from some of his patients. Until such pathologic studies can be made the definitive anatomic localization for the genetic trait will be in doubt. Such studies will be of the utmost value in furthering our knowledge of cochlear and cerebral cortical localizations of sound pathways. We have had too little opportunity to study the temporal bones and brains of individuals afflicted with perceptive deafness.

It is to be hoped that otolaryngologists will be on the alert and secure temporal bones and brains from any of their patients upon whom accurate audiometric records are available. The availability of audiometric studies on patients is only recent in otology. It is only during the past ten or fifteen years that fairly routine audiometric studies have been made on patients presenting the complaint of hearing loss. It is therefore to be hoped that in the next decade or two we will begin to accumulate sufficient temporal bone and brain material to correlate known audiometric information. "Nerve deafness," among other otologic terms, will then be re-evaluated and reclassified etiologically, anatomically, and pathologically.

I should like to congratulate Dr. Dolowitz on an important contribution to otology.

FRANZ ALTMANN, M.D., New York, N. Y.: The very interesting studies of Dolowitz and Stephens showed that many members of two kindreds suffered from a progressive nerve deafness which started as high tone loss in the second decade of life.

Anatomic investigations on this by no means rare type of hearing loss are, unfortunately, completely lacking. If it really represents a "premature aging of the hearing mechanism," the underlying anatomic changes should be similar to those in presbycusis. They would then consist mainly and primarily in degenerative changes in the cells of the spiral ganglion of the cochlea, starting in the lower half of the basal turn without involvement of the organ of Corti.

The findings in cases of so-called inherited deaf-mutism in which very marked or even complete deafness was already present at birth cannot satisfactorily explain a hearing loss which does not become manifest before the second decade of life and progresses only slowly.

This is particularly true of the findings in certain cases with a dominant hereditary trait where the cochlea is malformed (so-called *typus mondini*). The cochlear duct shows defective development and hydropic dilatation, particularly in the upper part, together with defects in the interscalar septa. If in these cases hearing is present at all, it is considerably reduced and more so in the lower than in the higher tonal range.

The known cases with a recessive hereditary trait show histologically degenerative atrophic changes in the epithelial structures throughout the cochlear duct, particularly of the organ of Corti and the stria vascularis. These changes may or may not be combined with atrophy of the spiral ganglion and the higher acoustic centers.

Of great interest are the findings in certain rodents with recessive deafness and locomotor anomalies. Although most of them become deaf shortly after birth, in some, the deafness starts between the twenty-second and thirtieth days, and in another mutation even much later, not before the fourth month of life. The histologic changes are of a similar type to those in man but less marked. But even in these rodents the deafness progresses very rapidly and leads in a short time to complete deafness.

It is, therefore, very improbable that primary changes in the stria vascularis and the organ of Corti could cause a slowly progressive deafness of late onset.

Premature aging of the hearing mechanism is, therefore, still the best explanation of the hearing loss in question. As in a number of other conditions, what is inherited is not a gross anatomic anomaly but a "localized organ weakness" (Streeter) which manifests itself in a lowered resistance of the cells of the spiral ganglion to ordinary wear and tear. This leads to an unusually early onset of those degenerative changes which are generally regarded as a characteristic feature of advanced age. The marked individual differences in the effect of prolonged exposure to loud sounds and in the time of onset and pace of progress of ordinary presbycusis are explained in a similar way.

Without discussing any of the genetic aspects of the cases, I just want to point out that recessive deafness is not infrequently combined with other inherited defects, for instance, with the recessive form of retinitis pigmentosa.

DR. DOLOWITZ: I wish to thank the two gentlemen for their discussions and for the encouragement they gave me when they received the paper.

As far as we can tell, we have been unable to find any other associated hereditary defect in these two families than those presented. One family is absolutely normal. The other has only the pyelonephritis as an associated difficulty.

I also am looking forward to the time when we can get an autopsy, but it is kind of hard to wish that on some of your patients.

IMPROVED HEARING IN CONGENITAL AURICULAR MALFORMATIONS: A REVIEW OF THE RESULTS OBTAINED BY SURGICAL METHODS

GEORGE L. PATTEE, M.D.
DENVER, COLO.

THE material for this paper was compiled from questionnaires sent to the members of the American Academy of Ophthalmology and Otolaryngology, and I want to thank all of you who were kind enough to send in the data on your cases. I realize that there are many gaps still to be filled in because, as is always the case, many reports were incomplete. Since none of us have enough of these cases to allow us to try various surgical technics, I hope that the information presented here will help to standardize the surgical technic to be employed in these cases.

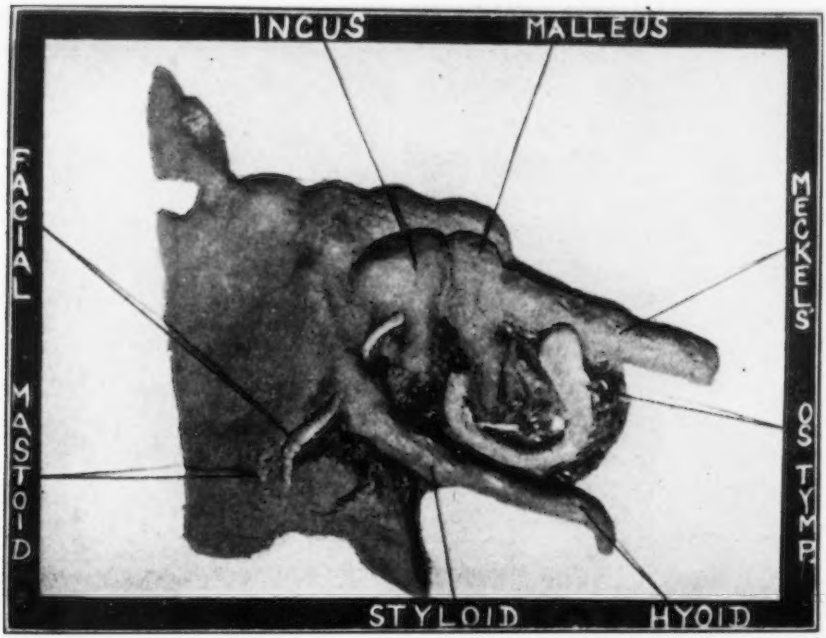

FIG. 1—Model to show the development of the ossicles, styloid and os tympanum from the first and second visceral arches. Modeled after Kollmann. (From Richards.[11])

The problem presented by these patients with congenital malformations of the middle and external ear is the same today as it was in 1914 when Dr. John Randolph Page[9] wrote as follows:

From the Department of Surgery, Division of Otolaryngology, University of Colorado Medical School.
Presented at the Fifty-Sixth Annual Session of the American Academy of Ophthalmology and Otolaryngology, Oct. 14-19, 1951, Chicago, Ill.

The attitude of writers on this subject in the past has been, with few exceptions, so universally against operation [redacted] without doubt a considerable number of skillful surgeons have been influenced by it to refrain from operation on cases that could have been materially helped. . . . [Such cases] are sufficiently numerous to warrant some attention being directed to them, particularly when it is found, as I believe investigation will show, that it is the rare exceptions where improvement in hearing will not be gained by the proper surgical procedure. The possibility of gaining for the child the early development of speech and expression and all that goes with improvement in hearing is well worth striving for.

unfortunate individuals, not to improve their appearance but to improve their hearing.

Time does not permit a presentation of the various types of embryologic defects encountered, but any one who intends to operate on this type of anomaly should first read the articles by Altmann,[1,2] Fraser[4] and Richards.[11] Dr. Richards has been kind enough to allow me to show pictures of his wax models which demonstrate the general nature of the anomaly.

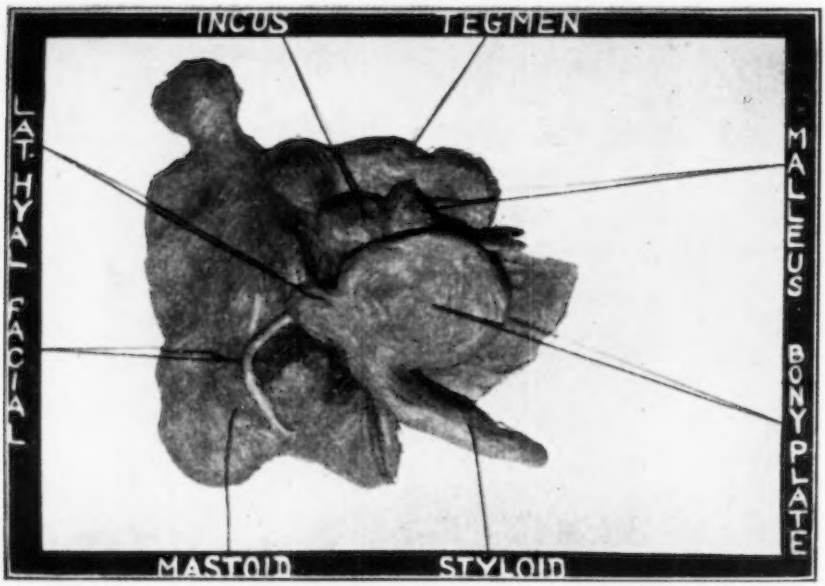

FIG. 2—Model to show the formation of the occluding bony plate from the laterohyale. Note absence of the os tympanum. Modified after Kollmann. (From Richards.[11])

In spite of Dr. Page's pleas for doing something for these patients with bilateral osseous atresia of the auditory canal, the attitude of otologists in general has been to leave them alone. Now because of a better understanding of the embryology of the congenital deformity and improvements in otological surgical technics, there has been a revival of interest in operating on these

The normal embryologic development is shown in figure 1. Please notice that the incus and malleus are formed from Meckel's cartilage, the stapes from the stylohyoid cartilage, and the tympanic bone is a separate and distinct anlage. In congenital atresia (fig. 2) notice that the tympanic bone which normally forms the auditory canal is absent. In its place is an enlargement of the stylo-

hyoid cartilage, which forms a bony plate. This bony plate occupies the normal position of the annulus and membrana tympani, and the incus and malleus may be attached to it. This bony plate may remain thin, or it may enlarge until it fills the entire space usually occupied by the auditory canal. In figure 3 the external appearance of a normal infant temporal bone and its relationship to the mandible are compared to that found in congenital atresia. Notice that there is no auditory canal present; allowance must be made for this absence of the auditory canal when starting to operate. In other words, during the operation what appears to be the posterior border of the auditory canal is actually the anterior surface which articulates with the mandible.

FIG. 3—Models to show the appearance of the normal external bony meatus and the same region in the presence of a congenital bony atresia. (From Richards.[11])

Because the auditory canal is never formed in these cases, Dr. Charles E. Kinney[6] of Cleveland believes that we should change our nomenclature and not call these cases congenital atresia. He believes that "atresia" implies that the canal was once formed and then closed, whereas in reality the canal never was formed.

After studying the data obtained, it is evident that each case presents special problems which are the results of the variation in the nature of the anomaly. The deformity of the auricle varies from normal to an almost complete absence. The canal may be occluded by a bony plate which is very thin and, in part, even fibrous, or it may be occluded by bone throughout its entire length. In a few cases, the occlusion is membranous and a normal membrana tympani is found. The incus and malleus may be normal, deformed, or absent. Often they are fused together as a single ossicle. The stapes is not deformed as frequently as the other ossicles, but it may be rudimentary, and even fixed by bony spicules, as shown by Kelemen (fig. 4).[5]

As a rule, either the incus or malleus articulates with and may adhere to the bony plate occluding the auditory canal. They may also be attached to abnormal bony spicules or fixed by fibrous bands to other portions of the middle ear cavity. The stapes may be fixed directly by bone spicules or indirectly by the fixation of the incus and malleus.

The facial nerve is often found in abnormal positions and in 2 cases it was reported as being found on the external surface of the mastoid process. Facial paralysis was reported as a complication in 4 cases, all of which showed recovery. I know that it has occurred in several other cases, but since the data were not submitted on these cases, they are not included.

One abnormality that is encountered rather frequently is the complete lack of pneumatization of the temporal bone. This defect has not received much attention previously because its importance has been overlooked. Fortunately it can be detected before surgery by means of roentgenograms so that the otologist can be prepared for the consequences. I have been unfortunate enough to operate on one of these sclerotic cases, and since there are absolutely no landmarks other than the dura of the middle fossa, the operation is most difficult. Of the 75 ears on which data were obtained, 6, or 8.0 per cent, showed this anomaly. In 3 ears which were operated on by very capable otologists, the operation could not be completed because the middle ear cavity was never found.

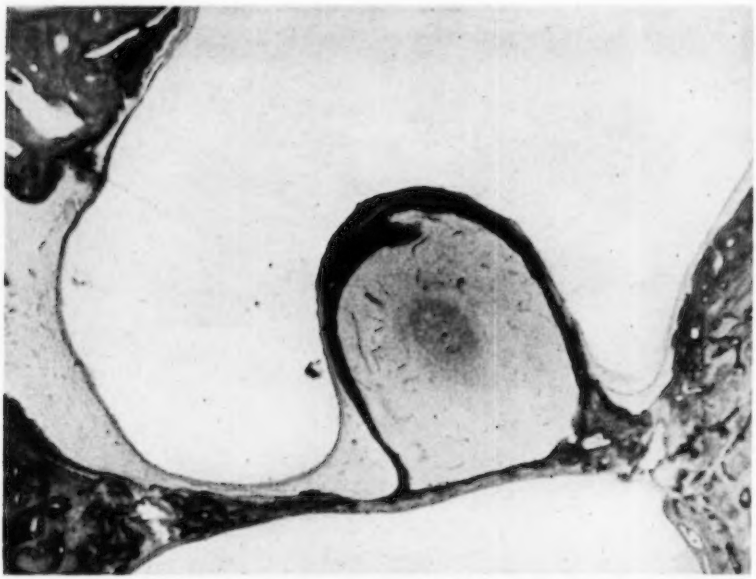

FIG. 4.—Deformed stapes, showing the crura only above the medial half of the footplate. The space between the crura is filled with connective tissue. There is no capitulum. The rudimentary stapedius muscle is without a tendon. (From Kelemen.[5])

Questionnaires were completed by 31 otologists representing their findings in 64 patients. There were 29 bilateral cases and 33 unilateral ones. In the unilateral cases the defect was found equally on both sides. As a point of interest, I have seen identical twins, one of whom

had the defect in the right ear and the other in the left ear. The sex ratio was 37 males to 25 females and almost all of this variation was found in the unilateral cases. This might be due to the fact that boys cannot hide their anomaly as well as girls and hence are more apt to seek surgery. Counting the bilateral operations, data were obtained on a total of 75 ears.

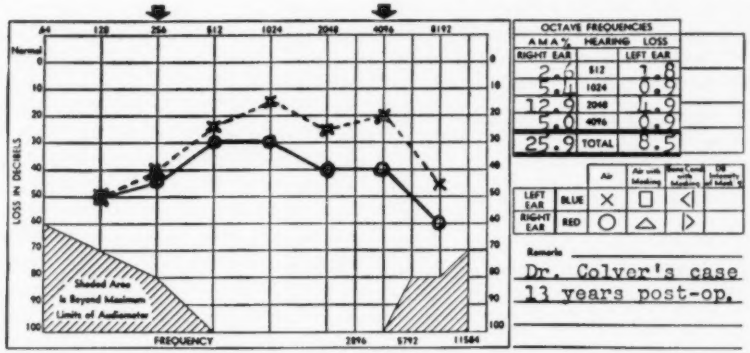

FIGURE 5.

Three otologists sent in data on patients that were operated upon prior to the publication of my paper[10] on this subject in 1947. The earliest operation on which follow-up audiograms are available was reported by Dr. Benton N. Colver[3] in 1933. As seen in figure 5, useful hearing has been maintained for 13 years after operation. Dr. Colver intends to re-present this case next spring at the meeting of the American Laryngological, Rhinological and Otological Society, so I will not discuss it further.

Altogether, audiograms were available for study on 47 ears. In these 47 ears, hearing was improved by surgery in 39, or 83 per cent, but the improvement was later lost in 6 ears so that permanent improvement in hearing occurred in only 33, or 70.2 per cent. However, in only 16 ears, or 34 per cent, did the hearing improve to the serviceable level of an average loss of 30 decibels or less in the critical speech frequencies.

The surgical procedures that have been carried out on these cases fall into several broad classifications. In the first group are found those surgical attempts to construct an auricle or an auditory canal for cosmetic reasons, without any attempt to improve hearing. Such cases are not included in this discussion. A second group that is not included consists of those cases in which the atresia of the auditory canal was not bony, but membranous. In these cases a normal tympanic membrane was found and the hearing returned to normal after the membranous obstruction of the canal was corrected. In the third group are found those cases in which the incus and malleus were removed in order to release the fixation of the stapes. In the fourth group an operation on the middle ear and mastoid was done, but none of the ossicles were removed. In the fifth group a fenestra was made in the labyrinth to improve the hearing.

In the third group there were 42 ears in which either the incus, malleus, or both were removed, and of these, 38, or 90.5 per cent, showed improvement in hearing. However, in 4, or 10.5 per cent, of the 38 ears in which the hearing improved, it later became worse, indicating that adhesions and scarring had caused

secondary fixation of the stapes after the operation. Audiograms were obtained in only 36 of these 42 ears. Only 11, or 30.6 per cent, had an improvement in hearing to an average loss of 30 decibels or less in the critical speech frequencies. Many of the patients and otologists were pleased with the results even though the hearing loss remained below the serviceable level of 30 decibels. However, one otologist reported that his patient still wears a hearing aid after the operation even though the average hearing loss was only 20 decibels.

Dr. Philip Meltzer[7] suggested that after the incus and malleus were removed, enough of the bony plate occluding the auditory canal should be removed so that the head of the stapes would be exposed. A conical skin graft should then be placed against the head of the stapes to create a much better sound-conducting mechanism.

A subgrouping of the third group of 42 ears in which the incus and malleus were removed was made in order to compare the results obtained by placing such a graft directly over the stapes with the results obtained when the graft was not against the stapes. There were 17 ears in this subgrouping and 16, or 94.1 per cent, showed improvement in hearing after the operations. One of the 4 ears which showed improvement in hearing which was lost subsequently was in this subgrouping. Audiograms were obtained on 14 ears in the subgroup and only 5, or 35.7 per cent, had an average serviceable level of hearing of 30 decibels or less in the critical speech frequencies. This compares with 6 ears out of 22, or 27.3 per cent, in which the average hearing loss was 30 decibels or less in the group where the graft was not placed against the stapes. The distribution of the hearing loss below the 30 decibel level was about equal in the two subdivisions. Thus it would appear from the data available that some advantage is to be gained by placing the graft over the stapes.

In the fourth group of 16 ears in which none of the ossicles were removed, 12, or 75 per cent, showed improvement in hearing following surgery. Two of the 12 ears, or 16.7 per cent, that showed an initial improvement in hearing lost it subsequently. Audiograms were obtained on 11 of these 16 ears, and 5, or 45.5 per cent, showed an average serviceable hearing level of 30 decibels or less in the critical speech frequencies. This group represents the ears in which the ossicles showed little if any malformation, and the occlusion of the auditory canal was sometimes a combination of bone and fibrous tissue. From studying the reports submitted, it is my belief that the increase in good results in this group was due to the mildness of the deformity rather than to the fact that the ossicles were left intact. A comment by one of our better otologists about his case in which the hearing improved and then worsened is of interest in this connection. He states:

> I had been mistaken in thinking that when I separated the bony attachment of the incus to the bony plate of the occluded canal that a movable ossicular chain would conduct sound with a full thickness graft over it. Subsequent experience showed I should have removed the incus and malleus.

Since the improvement in hearing following surgery is not consistent and frequently is not up to a serviceable level, fenestration of the labyrinth has been done in a few cases. This was first suggested by Ombredanne[8] who did it as a one-stage operation. In this country fenestration has been done both at the original operation and also as a subsequent procedure. Dr. De Graaf Woodman[12] recently presented a paper on a two-stage operation with fenestration in these cases, and since he is discussing this paper, I hope he will present his technic in more detail than my time will allow.

Data were received on 7 ears in which a fenestra had been made. In 5 of these the fenestra was made at the original operation, and in only 1 ear did the hearing improve to an average level of 30 decibels or less. The 2 cases in which the fenestra was made as a secondary operation are of sufficient importance to receive special attention. In one case the hearing improved after the first operation, but only to an average loss of 48 decibels. Following fenestration the hearing was further improved to an average loss of 20 decibels in the critical speech frequencies. In the other case, bilateral operations were done but there was no appreciable improvement in hearing in either ear, the average loss being 70 decibels. Subsequently a fenestration operation was done on one ear and the average hearing level improved to 37 decibels.

The only complications reported in this series of cases were facial paralysis, labyrinthitis from a one-stage fenestration, and secondary atresia of the auditory canal. As has been pointed out, the facial nerve often is in an abnormal position and the surgeon must be constantly on the alert to prevent injury to it. The incidence of labyrinthitis can probably be reduced if the fenestra is made at a second operation. The secondary atresia is troublesome and may require a subsequent operation. I believe it results because the new canal that is formed is not made as large as possible and the graft used is too small to cover the raw surface of the wound completely. It requires a piece of skin at least 2 inches in diameter to line the cavity properly, and it is far better to have too much than too little.

SUMMARY AND CONCLUSIONS

After studying the data submitted, I am of the opinion that all bilateral cases of congenital atresia of the auditory canal should be operated upon in an attempt to improve hearing. As a result of all the various types of surgical procedures used, a permanent improvement in hearing can be expected in about 70 per cent of the cases. This improvement in hearing is sufficient to bring the hearing up to an average loss of 30 decibels or less in the critical speech frequencies in only 34 per cent of the cases. Therefore, I believe that the first operation should be done in such a manner that a secondary fenestration operation as described by Woodman[12] can be done in those cases where the hearing fails to reach the serviceable level. This means that the incus and malleus should be removed and the labyrinth skeletonized, so that at the secondary operation it is only necessary to elevate the graft and make the fenestra.

The advisability of operating on the unilateral case is still debatable. As far as the hearing is concerned, the operation is not indicated as long as serviceable hearing is present in the normal ear. The only reason, then, for operating upon a unilateral deformity is to improve the appearance and perhaps thereby obtain a psychological improvement in the patient. In some cases these results have been of immeasurable value.

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DISCUSSION

GEORGE E. SHAMBAUGH, JR., M.D., Chicago, Ill.: The year 1947 is a milestone in the surgical correction of congenital atresia of the ear, for in that year independently and almost simultaneously Pattee of Denver and Ombredanne of Paris published the first really encouraging results.

To Dr. Pattee's very complete and very fine survey I should like to add and emphasize a few points.

First I wish to call attention again to the marked distortions of anatomy in the congenitally deformed outer and middle ear. The antrum may be very small or absent and the facial nerve may run an aberrant course. Facial paralysis is a very real hazard from this operation. The usual approach to the antrum is particularly dangerous, for the surgeon searching for an antrum that doesn't exist easily injures the facial nerve. Facial nerve paralysis has probably occurred more often than the statistics would indicate in operations for congenital atresia. It is well to recall that the first recorded operation for atresia by Kiesselbach in 1883 resulted in facial paralysis. No otologist should attempt this work who is not thoroughly trained and experienced in endaural surgery.

The second point is that Lempert's endaural approach is ideally suited for these operations. The danger of injury to the facial nerve can be minimized by using the atticotomy approach. By this we mean to open the attic first by removing the bone of the superior meatal wall until the attic is entered. Since in congenital atresia the osseous meatus is partially or entirely absent and the head of the mandible lies close to the mastoid process, we must remove the bone of the posterosuperior nonarticular portion of the temporomandibular fossa to reach the attic. Once the attic has been entered and the facial nerve has been identified, the cavity can be enlarged safely to the desired size under magnification with the binocular loupe.

The third point that I wish to make is that the same technic is not applicable to every case. Rather we must adapt the technic to the particular deformity that is found so as to construct a sound conducting apparatus that will most closely approximate the normal. Let me illustrate with a few examples.

The external auditory meatus begins in the embryo as a funnel-shaped depression representing the first branchial cleft. Later on, from the medial end of this "primary meatus" a solid cord of epithelial cells extends inward toward the tympanic cavity. Canalization of this solid cord of cells begins at the tympanic end to form the osseous or "secondary meatus," the canalizing process extending laterally to join the primary meatus and form the completed external auditory canal. If the arrest of development occurred just after the canalization began but before it was completed, there will be an atresia with a normal tympanic membrane ossicular chain. One needs only to create a large osseous meatus and line it with split thickness grafts to produce a normal sound conducting system. This was done in two ears in our series of 18 operations for congenital anomalies of the outer and middle ear.

The middle ear cavity comes from the first branchial cleft, while the cartilage of the first branchial arch, called Meckel's cartilage, forms the incus, malleus and mandible, and the cartilage of the second arch, called Reichert's cartilage, forms the incus. If Reichert's cartilage is the seat of the deformity, congenital stapes ankylosis may result with normal meatus, tympanic membrane and malleus and incus. Three of our ears showed this deformity and were fenestrated.

The most common deformity involves Meckel's cartilage with atresia, absent tympanic membrane, and deformed incus, malleus and mandible. The so-called Treacher-Collins syndrome with atresia and an "Andy Gump" jaw is the result. In these cases a new meatus lined by Thiersch grafts is created, the deformed incus and malleus are removed and a full thickness graft is placed against the stapes to form a substitute tympanic membrane. This was done in five of our ears. If the malleus and incus appear normally mobile, we have left them in place and have placed the full thickness skin graft against them. We are not yet sure that this is better than placing the graft against the stapes, but theoretically the sound conducting system is nearer normal with an ossicular chain. We do not believe the labyrinth should be fenestrated routinely as advocated by Ombredanne, but only when there is stapes ankylosis.

The hearing results in our series of 18 operated ears are similar to those reported by Dr. Pattee; actually most of our cases are included in his series. At the most recent test, 11 of our 18 ears were at the level of practical hearing.

In one of our cases no middle ear attic or antrum could be found, and the operation was not completed. Except for one patient with a facial paralysis operated upon elsewhere by the postaural route who came to us for repair of the nerve, there have been no cases of facial nerve paresis or paralysis. I agree with Dr. Pattee that in selected cases with good cochlear function the operation for congenital atresia by the endaural route is a useful procedure which offers the patient a good chance of gaining practical hearing.

DE GRAAF WOODMAN, M.D., New York, N. Y.: Before showing the slides of the two-stage technic with fenestration I should like to ask Dr. Pattee one question. Did he record the time factor between the operative procedure and the taking of the audiograms? The reason I mention this is because in the case that we did following the first-stage operation, which was done much according to Pattee's technic, we had a remarkable result. A 15 or 20 decibel level was obtained and this held up until about the tenth week. Then in the third month it dropped down to 48 decibels and there it stayed. If this factor was not recorded, it might make some difference in the averages which were found. The same thing holds true for a fenestration operation. We generally do not consider we have a good result until at least one year has elapsed.

(Slide) This case shows a minimal amount of deformity of the right ear; therefore, we chose it for the side to operate upon.

(Slide) The preoperative audiogram shows excellent bone conduction across the board and an average of 50 to 60 decibels in the air conduction.

(Slide) The first-stage procedure was carried out by an endaural approach, and, as you can see, when we got down to the cortex there was no sign of a canal or any landmarks to go by. In this particular case we found that he had a fairly well-pneumatized mastoid, so at least we had hope we were going to find something under that cortex.

(Slide) After getting through the cortex and going down and finding the middle ear, we encountered an atrophic incus and malleus. The stapes was found to be well developed and mobile. From then on the procedure was to skeletonize the horizontal canal thoroughly in order to prepare it for fenestration, should a second-stage operation be found necessary. A dermatome skin graft was then made from the abdomen and it took 4 square inches. We take considerably more than we think necessary. It looks like an enormous cavity at this stage of the game. This was sectioned into several pieces, and one piece was placed into the depth of the cavity, covering the canals and the middle ear. The other pieces were attached to the periphery of the wound and allowed to drop down into the cavity. The cavity was then filled with melted paraffin. One week later the melted paraffin was removed and a duplicate acrylic mold was made from this, through which a quarter-inch hole was drilled in order to allow drainage. Then this mold was used during the epithelization stage in order to help keep the cavity from contracting down too much, for even with this procedure the cavity becomes relatively small.

(Slide) This shows the ear and the canal following the operative procedure. This two-stage technic permits of obtaining a viable flap with a minimal amount of tissue reaction and, therefore, very little opportunity for secondary infection, with the hazard of developing labyrinthitis and loss of cochlear function.

(Slide) In this second stage all that remains to be done is to reopen by an endaural incision and go down and cut a flap. The flap is cut in such a manner that the hinge is placed anterior to the horizontal canal. The flap was elevated readily. There was little or no bleeding, and the fenestration was done in the usual manner and the flap placed back in position and held in place with light paraffin mesh gauze.

(Slide) The dotted line represents the preoperative audiogram. The single line represents the final drop of the audiogram four months after the first procedure. You see it originally was way up around 15 or 20 decibels, but that lasted only about ten weeks and then it dropped down to the 48 decibel level. The dotted line represents the audiogram following fenestration. That was taken at six months. I took one a week ago which represents seventeen months postfenestration and it remains up in that same level.

CHARLES E. KINNEY, M.D., Cleveland, Ohio: I should only like to add that in my mind this is one of the more monumental pieces of work that have been done in our field of otology. I did have a facial paralysis in one of my cases. Fortunately, it has recovered approximately 80 per cent. It was in the type of case that Dr. Shambaugh referred to, the so-called Treacher-Collins syndrome, and I should like to make this addition. In those cases, the temporal muscle is completely haywire. The facial paralysis resulted when I nearly completely severed the facial nerve on my incision, before I had done a bit of bone work. When we reoperated upon the case three weeks later we found a nearly severed facial nerve at the upper end of the endaural incision. To repeat, it was at the upper end of the endaural incision. The point of exit of the facial nerve was approximately 7 millimeters cephalad to the temporomandibular joint. It is in those cases that one finds the greatest abnormalities in the placement of the facial nerve.

DR. PATTEE: I want to thank Dr. Kinney, Dr. Woodman, and Dr. Shambaugh for their kindness in their discussion.

Dr. Woodman asked me whether or not particular attention was paid to the length of time after surgery that the audiograms were reported. The questionnaires asked this but it was not always answered, and so it was not always taken into consideration. However, as I reported, there was an immediate improvement in hearing in some patients which later worsened.

Dr. Fowler asked me at what age I would advise doing this operation. That is still debatable perhaps, but I am sure that any of us who heard Mrs. Tracy Sunday night would agree that the sooner you can improve the patient's hearing to a serviceable level the better. However, as I stated in 1947, I do not advocate operating on these individuals until you are sure there is hearing present, because in some cases the defect is such that there is no hearing present, and in those cases there is no use in operating.

One other thing should be added, after seeing Dr. Peluse's pictures yesterday. In the past I have felt that prostheses made of rubber and painted were not satisfactory in these cases. I am certainly interested in the new plastic prostheses that he showed us, and if they will work, I would much rather see a unilateral case wear a prosthesis than have it operated on.

TRANSACTIONS

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RETINAL DETACHMENT

A TIMELY summation of the researches carried out in a number of institutions by diagnosticians and surgeons on the causes and cures of retinal detachment, as presented at the 1951 meeting of the Academy, appears in this issue of the TRANSACTIONS. The Program Committee went all overboard to gather the most authoritative information possible and to present it in the most practical condensed form.

The symposium was conceived by the Secretary for Ophthalmology, Dr. Algernon B. Reese, and delivered by the Program Committee to Dr. Lawrence T. Post with instructions to nurse and direct the progress of the prodigy more than two years before the anticipated debut. The presentation at the meeting last October was not just a reading of unrelated papers on the subject by a number of authors but a carefully worked out plan covering the entire clinical aspect of retinal detachment. The participants in the symposium met several times in the two years of preparation of the report, and under the superb direction of Dr. Post, the voluminous material, published and unpublished, made available by masters in the field was marshalled and condensed and fitted into a body of magnificent proportions. Fellows who witnessed the presentation should know that months of studied effort by the authors went into the preparation of the text, and the presentation itself was completely rehearsed the night before. The publication of a wealth of illustrations in color was necessitated by the nature of the diagnostic distinctions brought out by the authors, that could be only inadequately demonstrated without color.

We were particularly fortunate in having as Guest of Honor of the meeting, Dr. Hermenegildo Arruga, whose contribution to the program on "Certain Considerations of the Surgical Treatment of Retinal Detachment" complemented the symposium by our own members. Dr. Arruga's paper with thirty-four illustrations in color will appear in the July-August issue of the TRANSACTIONS.

The combined papers of Dr. Post's symposium and of Dr. Arruga constitute a text-atlas on retinal detachment that is unequaled. They will be made available for future ophthalmologists in the form of a manual, of the series prepared for the Home Study Courses.

Probably the last word on retinal detachment has not been said. Extensive researches are in progress in a number of cities. Etiological factors may be

found to differ in various parts of the world; new methods of determining the presence and extent of flat detachments may be demonstrated; new methods for cure may be discovered, but the ophthalmoscopic appearance of the condition has been fully depicted. That contribution is basic and not subject to change. It has been recorded in the best possible manner and made available to everyone through the pages of the TRANSACTIONS.

WILLIAM L. BENEDICT, M.D.

MOTION PICTURES

THE Academy last year collected the essential data on 72 motion pictures within its several subspecialties. This preliminary survey was carried out in conjunction with the Medical Audio-Visual Institute of the Association of American Medical Colleges as a part of its over-all catalog reference card project being developed with the Library of Congress. The 72 film titles are listed elsewhere in the TRANSACTIONS and represent a limited coverage of the very large number of films produced for all purposes by the Academy membership. Large blocks of films are obviously missing. Therefore, in order to have adequate coverage for real reference purposes, the Academy will supply on request additional copies of the form for completion. Newly produced films should be routinely reported to the Academy for inclusion in the Academy and Association's files and bulletins.

An analysis of the forms submitted not only underlines the incompleteness of the returns but also poses certain questions. Since motion pictures are being increasingly used in graduate instruction in the specialties, each film author must answer two questions: Will he make his film available to others on suitable request? And if he will, is he prepared to distribute the film as a print, which means additional cost, or to risk destruction of the original through sheer wear or through improper projection? The Academy is not now prepared to undertake methodical distribution even of those films of high general interest. But the experience and reactions of the members will permit development of a sound Academy policy on motion pictures which can be integrated with the programs of other national agencies.

DIRECTIONS FOR CLEANING AND DISINFECTING TONOMETERS

THE Committee on Standardization of Tonometers has made a special study of the problem of cleaning and disinfecting tonometers and presents the recommendations listed below.

JONAS S. FRIEDENWALD, M.D.
Chairman

1. *Disinfection*

In order to avoid the transference of infectious bacteria or viruses from one patient to another, it is advisable to keep the end of the tonometer coming in contact with the eye in a solution of 1:5000 zephiran during the period of the day in which the tonometer is in use. In place of the zephiran an organic mercurial disinfectant may be employed. Suitable stands are now available on the market by means of which the tonometer may be kept ready for use with the footplate and the plunger immersed in such a disinfecting solution.* The instrument should not be left in the disinfecting solution overnight as corrosion of the metal may occur. Since the disinfectant may be slightly irritating to the eye, the footplate and lower end of the plunger should be washed with distilled water and wiped dry with a gauze or cellocotton sponge immediately be-

*Dr. Albert D. Ruedemann was the first to devise such a convenient stand and place it on the market (fig. 1).

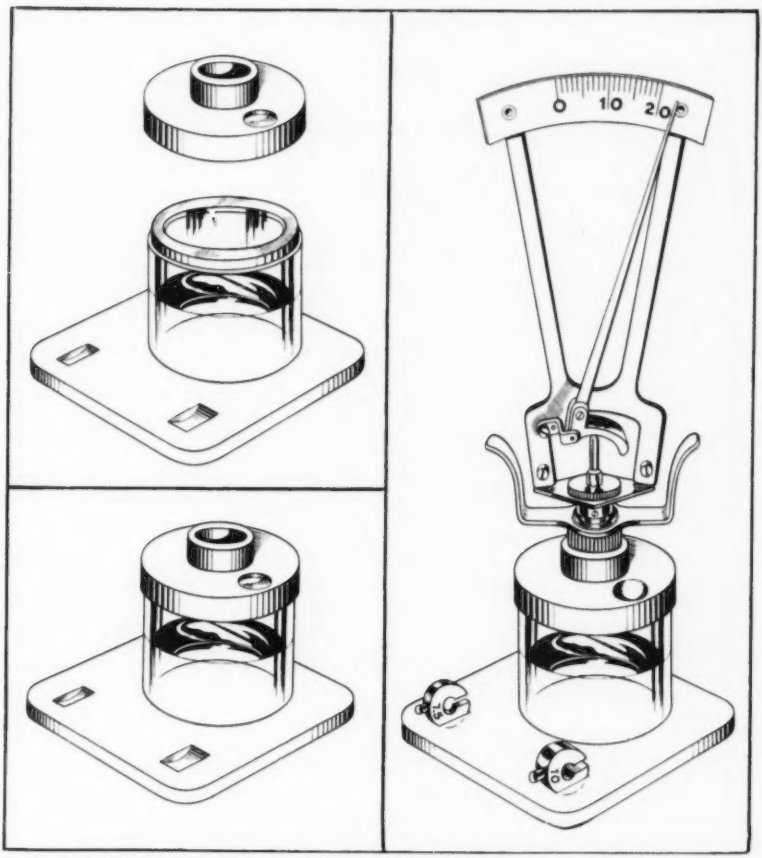

FIG. 1—Tonometer stand for disinfection devised by Dr. Albert D. Ruedemann.

fore use. If a suitable stand is not available, the tonometer footplate and lower end of the plunger should be wiped with a gauze or cotton sponge moistened with 1:5000 zephiran or an aqueous solution of one of the organic mercurial disinfectants before and after each use.

2. *Cleaning*

At the end of the day's use the plunger should be removed from the cylinder after removing the 5.5 gram weight. Wipe the plunger clean with a gauze sponge moistened with 70 to 95 per cent alcohol. Irrigate the plunger casing of the cylinder thoroughly with distilled water. It is preferable to have this warm in order to dissolve rapidly any crystals of salt which have formed from tear fluid. Following this, the casing should be irrigated with 70 to 95 per cent alcohol and the tonometer allowed to dry. The tonometer and the plunger should then be kept in the closed carrying case until the next day's use.

In Memoriam

Clyde Olen Brown.....St. Louis, Missouri
February 12, 1952

Charles Firestone.....Seattle, Washington
January 18, 1952

William Davis Gill.....San Antonio, Texas
May 24, 1952

Bernard Norman Gottlieb.....Brooklyn, New York
January 30, 1952

Louis Kauffman Guggenheim.....Los Angeles, California
March 13, 1952

Marvin Fisher Jones.....New York, New York
May 26, 1952

Melchiore Lombardo.....Brooklyn, New York
February 10, 1952

William Henry Luedde.....St. Louis, Missouri
March 19, 1952

John Ambrose Monahan.....Clinton, Massachusetts
February 24, 1952

William Wallace Ralston.....Houston, Texas
November 3, 1951

Jacob Rosenbaum.....Montreal, Quebec, Canada
April 26, 1952

James McMahon Schmidt.....Brooklyn, New York
May 18, 1952

Richard Gordon Scobee.....St. Louis, Missouri
June 22, 1952

William Lawrence Sheahan.....New Haven, Connecticut
December 16, 1951

Thomas Gooch Tickle.....New York, New York
April 26, 1952

Helen Frances Upham.....Asbury Park, New Jersey
February 3, 1952

FINANCIAL REPORT

Annual Audit for Calendar Year — 1951

May 15, 1952

The American Academy of
Ophthalmology and Otolaryngology
Rochester, Minnesota

Gentlemen:

As requested, we have examined the balance sheet of the American Academy of Ophthalmology and Otolaryngology, an unincorporated association, Rochester, Minnesota, for the year ended December 31, 1951, and the related statements of operations and net worth for the year then ended. Our examination was made in accordance with generally accepted auditing standards, and included such tests of the accounting records and such other auditing procedures as we deemed necessary in the circumstances. We submit herewith our report which includes the following statements:

Exhibit

"A"—Balance Sheet, December 31, 1951
"B"—Statement of Operations, Year Ended December 31, 1951
"C"—Analysis of Undisbursed Funds from Special Contributions
"D"—Analysis of Net Worth Account
"E"—Analysis of Increase in Market Value of Securities

Schedule

1—Securities Owned, December 31, 1951

Current Assets

Cash on deposit at the various banks was verified by communicating with the depositories, the funds in the Canadian bank being stated in U. S. dollars. The petty cash was verified by actual count.

Membership dues receivable, amounting to $1,108.00, consists of delinquent dues for 1951 and prior years which were verified from the records only. Other receivables totaling $1,723.23 consists of $1,100.00 due for films and slides and $623.23 due from members and others for services, abstracts, manuals, etc.

Inventory of abstracts, manuals, bound volumes, films and slides, in the amount of $15,498.13, was taken from the inventory records without further verification except as to tests of prices, extensions and footings.

Investments

Investments in bonds and stocks, which are described in Schedule No. 1, had a market value of $242,854.51 on December 31, 1951, as compared with $240,089.63 on December 31, 1950. U. S. Savings Series G bonds are stated at maturity values rather than current redemption prices.

Securities in the "Educational Fund," which are held for safekeeping by the Trust Department of the First National Bank, Minneapolis, Minnesota, were verified by communicating with the custodian of the securities. U. S. Savings Series G bonds in the "Wherry Memorial Fund" are held by Dr. W. L. Benedict, Executive Secretary-Treasurer, and were inspected by us. Interest earned on bonds since the dates on which last payments became due, has not been accrued. An analysis of the increase in the market value during the year is shown in Exhibit "E."

Fixed Assets

Furniture, fixtures and equipment amounting to $8,428.33 represents equipment acquired by the Academy over a period of years. Equipment purchased since September 1, 1942, amounting to $7,008.05 was for the most part verified through an inspection of supporting vouchers, and of the latter amount, $1,610.64 was purchased during the current year. Equipment acquired in years prior to September 1, 1942, which had been charged to operating expenses as purchased, was inventoried and appraised at a market value of $1,420.28 as of January 1, 1944, and set up on the books for that amount. Depreciation has been deducted and charged to operations at a rate that appears to be adequate.

Embryology motion picture film and slides, in the amount of $40,900.00, represents the cost to complete a medical motion picture film and slides on "Embryology of the Eye" by Sturgis-Grant Productions, Inc. The master films are being used to produce prints and slides and these are sold to those requesting them. The cost of the original film and slides is being amortized at a rate that appears to be adequate.

Deferred Charges

Unexpired insurance premiums as computed by us amounted to $410.25. Supplies in the amount of $303.50 represents stationary stock held by the respective manufacturers.

Prepaid postage deposit of $62.72, verified by correspondence, represents the balance of cash advanced to the U. S. Post Office at Omaha, Nebraska, to be applied as postage on subsequent mailings of the TRANSACTIONS and other periodicals by the publisher at Omaha.

The advance payment of $2,500.00 on a motion picture project represents the initial payment on a contract with Sturgis-Grant Productions, Inc., to produce a film, "Embryology of the Human Ear," at a total cost of $7,500.00 to cover script and story board only.

Current Liabilities

Accrued expenses totaling $5,085.11 represent unpaid December printing cost of the TRANSACTIONS and other current expenses. Membership dues represents amounts paid in advance by association members. Advance payments of $2,493.00 on bound volumes represents payments received as of December 31, 1951, for which volumes have not as yet been delivered.

Undisbursed Funds from Special Contributions

Undisbursed funds from contributions received and designated for "Specific Projects" totaling $8,425.05 are analyzed in Exhibit "C," which shows 1951 transactions as well as prior year balances.

Net Worth

An analysis of the net worth of the Academy is shown in Exhibit "D." Because the increase in market value of securities was greater than the loss from current operations, the net worth increased from $296,038.59 on December 31, 1950, to $298,138.35 on December 31, 1951.

OPERATIONS

A statement of operations for the year ended December 31, 1951, is presented in Exhibit "B." A summary of this statement and a comparison with the results for the year 1950 is as follows:

	Year 1951	*Year* 1950	*Increase* **Decrease**
Revenues	$172,681.10	$158,407.74	$14,273.36
Operating Expenses	173,558.72	137,623.37	35,935.35
Net Income - **Loss**	**877.62**	$ 20,784.37	**$21,661.99**

The decrease in net income from a year ago is due principally to increased general administrative expenses, the increased cost of publishing the TRANSACTIONS of the Academy and the publication of the Transactions and Directories of the first Pan-American Congress of Otolaryngology held October 17, 18 and 19, 1946.

GENERAL

Upon the examination of the insurance policies in force, we found Comprehensive General Liability insurance in the amount of $25,000.00 for each accident with a total limit of $25,000.00 and special limits of $50,000.00 to $100,000.00 covering the Chicago convention. Workmen's compensation insurance, which is being carried at the statutory amounts, now includes coverage for all persons employed at your annual convention.

The Executive Secretary-Treasurer and Chairman of the Finance Committee are bonded for $25,000.00 each. The principal office employees are bonded only for $1,000.00 each, which in our opinion is inadequate.

Fire and extended coverage on furniture, fixtures and equipment is carried at $7,500.00. Paper stock and publications in custody of the publisher at Omaha, Nebraska, are insured for $12,000.00. Office cash and securities are covered up to $2,400.00 within the premises and $2,100.00 outside the premises against safe burglary and messenger robbery. In view of the considerably larger sums of cash and securities handled by employees at convention time we believe that this coverage should be increased.

We acknowledge the very courteous assistance extended to us by Dr. W. L. Benedict, Executive Secretary-Treasurer of the Academy, and his staff during the progress of this examination.

OPINION

In our opinion, subject to the comments in the preceding paragraphs, the accompanying balance sheet and related statements of operations and net worth present fairly the financial position of the American Academy of Ophthalmology and Otolaryngology at December 31, 1951, and the results of its operations for the year then ended, in conformity with generally accepted accounting principles applied on a basis consistent with that of the preceding year.

Respectfully submitted,

BYERS, WOBSCHALL & MILLER
By Edw. A. Wobschall, C.P.A.

EXHIBIT "A"

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

Balance Sheet as at December 31, 1951

ASSETS

Current Assets:			
Cash, First National Bank, Rochester, Minnesota		$ 2,956.25	
Cash, First National Bank, Minneapolis, Minnesota		9,041.74	
Cash, Bank of Montreal, Toronto, Canada		1,301.66	
Petty cash fund		25.00	
Membership dues receivable, delinquent		1,108.00	
Accounts receivable, other		1,723.23	
Inventory, abstracts, manuals, monographs, films, etc.		15,498.13	
Total current assets			$ 31,654.01
Investments: (Schedule No. 1)			
Bonds and stocks			242,854.51
Fixed Assets:			
Furniture and fixtures	$ 8,428.33		
Less reserve for depreciation	4,336.17	4,092.16	
Embryology Motion Picture Film & Slides	40,900.00		
Less reserve for depreciation	7,760.00	33,140.00	37,232.16
Deferred Charges:			
Prepaid insurance		410.25	
Supplies		303.50	
Prepaid postage		62.72	
Embryology of the Human Ear Motion Picture		2,500.00	3,276.47
Total Assets			$315,017.15

LIABILITIES AND NET WORTH

Current Liabilities:		
Accrued expenses	$ 5,085.11	
Deferred income, membership dues, etc.	198.24	
Deferred income, advance payments on bound volumes	2,493.00	
Withholding tax payable	677.40	$ 8,453.75
Undisbursed Funds from Special Contributions (Exhibit "C")		
Wherry Memorial Fund	5,012.72	
Economic Survey of Impaired Hearing	957.46	
Sub-committee on Noise in Industry	2,429.87	
Isabelle Wilson Research & Education Fund	25.00	8,425.05
Net Worth (Exhibit "D")		298,138.35
Total Liabilities and Net Worth (*)		$315,017.15

* Subject to report comments

EXHIBIT "B"

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

Statement of Operations

Year Ended December 31, 1951

Revenues:			
Membership dues			$ 28,862.00
Membership applications			7,500.00
Abstracts, manuals and monographs			9,695.15
Certificates			1,409.50
TRANSACTIONS			40,008.91
Home Study Courses			4,234.00
Convention			66,189.20
Testing and calibration of tonometers			1,285.00
Films and slides			5,375.00
Income from securities:			
Interest, Educational Fund		$ 5,045.44	
Dividends, Educational Fund		2,106.46	7,151.90
Other Income			970.44
Total Revenues			172,681.10
Operating Expenses:			
General administrative		78,141.79	
TRANSACTIONS		35,400.88	
Home Study Courses		8,007.27	
Convention expenses		43,033.44	
Educational Fund:			
Supplements, etc.	$ 3,709.16		
Depreciation, Embryology Film & Slides	3,950.00	7,659.16	
Total Operating Expenses			172,242.54
Operating Income			438.56
Other Expenses:			
Loss on sale of securities			1,316.18
Net Loss			**$ 877.62**

EXHIBIT "C"

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

Analysis of Undisbursed Funds From Special Contributions

WHERRY MEMORIAL FUND	
Balance, December 31, 1950	$ 4,897.72
Receipts:	
Interest on U. S. Savings bonds, year 1951	115.00
Balance, December 31, 1951	$ 5,012.72
ECONOMIC SURVEY OF IMPAIRED HEARING FUND	
Balance, December 31, 1950	$ 6,200.00
Receipts: Contributions received in 1951	800.00
Total	7,000.00
Less: Expenditures during 1951	6,042.54
Balance, December 31, 1951	$ 957.46
SUB-COMMITTEE ON NOISE IN INDUSTRY FUND	
Balance, December 31, 1950	$ 567.51
Receipts: Contributions received in 1951	6,250.00
Total	6,817.51
Less: Expenditures during 1951	4,387.64
Balance, December 31, 1951	$ 2,429.87
ISABELLE WILSON RESEARCH & EDUCATION FUND	
Balance, December 31, 1950	$ 25.00
Receipts and expenditures in 1951	None
Balance, December 31, 1951	$ 25.00

EXHIBIT "D"

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

Analysis of Net Worth Account

For Year Ended December 31, 1951

Net Worth, December 31, 1950	$296,038.59
Additions:	
Increase in market value of securities during 1951 (Exhibit "E")	2,977.38
	299,015.97
Deductions:	
Net loss, year ended December 31, 1951 (Exhibit "B")	877.62
Net Worth, December 31, 1951	$298,138.35

EXHIBIT "E"

Analysis of Increase in Market Value of Securities

	Cost	*Market Value*	*Increase* **Decrease**
December 31, 1950	$241,450.98	$240,089.63	**$1,361.35**
December 31, 1951	241,238.48	242,854.51	1,616.03
Difference	**$ 212.50**	$ 2,764.88	$ 2,977.38

SCHEDULE No. 1

American Academy of Ophthalmology and Otolaryngology

Securities Owned, December 31, 1951

EDUCATIONAL FUND BONDS	RATE %	MATURITY	PAR VALUE	COST	MARKET VALUE *
Citizens Building Co. of Cleveland, 1st Leasehold Sinking Fund	6	1/1/46	$ 5,000.00	$ 5,000.00 (1)	None
Van Swerigen Co. Certificates of Indebtedness, Series 1925	6	12/31/48	6,000.00	3,477.03	$ 750.00
Dominion of Canada, 8th Victory Loan Ser. P5	3	10/1/63	2,500.00	2,500.00 (2)	2,321.88
Dominion of Canada, 8th Victory Loan Ser. P5	3	10/1/63	5,000.00	5,112.50 (2)	4,643.75
U.S.A. Savings, Series F	2½	12/1/55	13,500.00	9,990.00	11,799.00
U.S.A. Savings, Series G	2½	8/1/53	10,000.00	10,000.00 (3)	10,000.00
U.S.A. Savings, Series G	2½	6/1/55	4,000.00	4,000.00 (3)	4,000.00
U.S.A. Savings, Series G	2½	3/1/56	6,000.00	6,000.00 (3)	6,000.00
U.S.A. Savings, Series G	2½	5/1/56	8,000.00	8,000.00 (3)	8,000.00
U.S.A. Savings, Series G	2½	7/1/56	2,000.00	2,000.00 (3)	2,000.00
U.S.A. Savings, Series G	2½	11/1/56	25,000.00	25,000.00 (3)	25,000.00
U.S.A. Savings, Series G	2½	1/1/57	2,000.00	2,000.00 (3)	2,000.00
U.S.A. Savings, Series G	2½	7/1/58	30,000.00	30,000.00 (3)	30,000.00
U.S.A. Savings, Series G	2½	12/1/58	25,000.00	25,000.00 (3)	25,000.00
U.S.A. Savings, Series G	2½	3/1/59	15,000.00	15,000.00 (3)	15,000.00
U.S.A. Savings, Series G	2½	7/1/60	30,000.00	30,000.00 (3)	30,000.00
U.S.A. Treasury, 1966-71	2½	3/15/71	25,000.00	25,218.75	24,078.13
Total Bonds				$208,298.28	$200,592.76

STOCKS	KIND	PAR VALUE No Par	SHARES		
Citizens Building Co. of Cleveland	Shares		50	None	None
Commonwealth Edison Co.	Common	$25.00	100	$ 2,619.00	$ 3,050.00
E. I. duPont de Nemours & Co.	Common	5.00	60	2,347.31	5,520.00
Louisville Gas & Electric Co.	5% Pfd.	25.00	200	5,450.00	5,700.00
Massachusetts Investors Trust	Trust Cert.	1.00	617	17,923.89	23,291.75
Ophthalmic Publishing Co.	Capital	20.00	5	None	100.00
				$ 28,340.20	$ 37,661.75
Total Educational Fund				$236,638.48	$238,254.51

WHERRY MEMORIAL FUND	RATE	MATURITY	PAR VALUE		
U.S.A. Savings, Series G	2½%	1955	$4,600.00	$ 4,600.00	$ 4,600.00
Total Investments				$241,238.48	$242,854.51

(*) Except for U.S.A. Savings, Series G bonds, which are included at maturity values
(1) No market available
(2) U. S. dollars
(3) Maturity values

LIST OF MOTION PICTURES

LAST year, the American Academy of Ophthalmology and Otolaryngology mailed film questionnaires to its members. From the returns, the Medical Audio-Visual Institute of the Association of American Medical Colleges has compiled and edited the list below.

The information furnished by the authors or producers of the films varied in regard to completeness and detail. Several of the films have been omitted from this list because adequate data on them could not be obtained. Some other films were eliminated because their owners do not make them available to others, or use the original film for projection without having provided for the making of prints.

It is hoped that the present list can be expanded so that gradually a complete inventory of motion pictures in the field of ophthalmology and otorhinolaryngology will be assembled.

The listing of ophthalmologic films was edited by Adolf Nichtenhauser, M.D., and that of the others by David S. Ruhe, M.D.

All films listed are in the 16 mm. size.

OPHTHALMOLOGY

ANATOMY

The Zonule of Zinn and Its Chambers
Color, silent, 40 minutes.
Years of Production: 1940-46, revised 1950; **Author and Producer:** Henry Minsky, M.D.; **Camera:** Robert J. Carlin.
Distribution: Henry Minsky, M.D., 2 East 95th St., New York 28, N. Y., **Loan.**

Dissection of the eye to demonstrate the anatomy of the zonule of Zinn and of an aqueous, real space, the space of Garnier or zonular chamber, which can be injected by an opaque dye. Relationship of the lens to the vitreous body. Applied anatomy of the cataract operation. Suggested application to the physiology of accommodation and the mechanism of spontaneous detachment of the retina.

EMBRYOLOGY

Embryology of the Eye
Color, sound, 45 minutes.
Year of Production: 1950; **Sponsor:** American Academy of Ophthalmology and Otolaryngology; **Technical Adviser:** George K. Smelser, Ph.D.; **Producer:** Sturgis-Grant Productions, New York, in cooperation with Carnegie Institute of Washington, Department of Embryology.
Distribution: American Academy of Ophthalmology and Otolaryngology, 100 First Ave. Bldg., Rochester, Minn., **Sale:** $250; Committee on Medical Motion Pictures, American Medical Association, 535 North Dearborn St., Chicago 10, Ill., **Rental:** $3.

Part I presents the early stages, and Part II the later stages. The development of the lens, vascular system, cornea, lids, muscles, and retina is first shown simultaneously, and later separately and in detail for each organ structure.

MEDICAL OPHTHALMOLOGY

1. Glaucoma

Glaucoma; What the General Practitioner Should Know
Color, sound, 23 minutes.
Year of Production: 1951; **Sponsor:** National Society for the Prevention of Blindness; **Scientific Adviser:** National Society's Committee on Glaucoma; **Producer:** Sturgis-Grant Productions, Inc., New York, N. Y.
Distribution: National Society for the Prevention of Blindness, 1790 Broadway, New York 19, N. Y. **Sale:** $125.

A visual presentation of the mechanism of glaucoma, its early signs and symptoms, and the rationale of treatment.

2. Leprosy

Ocular Leprosy (Palo Seco Leprosarium, Panama)
Silent, 15 minutes.
Year of Production: 1945; **Author and Producer:** R. D. Harley, M.D.
Distribution: R. D. Harley, M.D., 101 South Indiana Ave., Atlantic City, N. J., (no data given).

Shows views of colony and of advanced cases with ocular complications.

REFRACTION

Errors of Refraction
Color, silent, 35 minutes.
Year of Production: 1944; **Sponsor:** Audio-Visual Medical Education Dept., Northwestern University Medical School; **Authors:** William A. Mann, M.D., and Emile G. Nadeau, Jr., M.D.; **Producer:** Mervin W. LaRue, Chicago.
Distribution: Audio-Visual Medical Education Dept., Northwestern University Medical

School, 303 East Chicago Ave., Chicago 11, Ill. **Sale:** $168; **Rental:** $5 per showing.

Presents the basic principles involved in optical properties of lenses and the refractive errors of the eye. Intended as an introduction.

EXTERNAL DISEASES

Trachoma

Trachoma in the Missouri Ozarks
Black-and-white, silent, 45 minutes.
Year of Production: 1938, revised 1950; **Author and Producer:** A. A. Siniscal, M.D.
Distribution: A. A. Siniscal, M.D., Missouri Trachoma Hospital, Rolla, Mo., **Loan.**

Animated drawings of eyeball and lids, showing formation of granules of trachoma, pannus, and entropion. Typical inhabitants of trachoma areas; the fight against control; field nurses and doctors visiting patients in the field, and examining and treating on the spot. Trachoma distribution among the States. Trachoma pathology (gross). Procedures of entropion and grattage surgery. Also for nurses and social workers.

SURGERY

1. Anesthesia

Deep Orbital Anesthesia to Produce Motor Block of the Extraocular Muscles
Color and black-and-white, silent, 20 minutes.
Year of Production: ? ; **Author and Producer:** Harold Gifford, M.D.
Distribution: Harold Gifford, M.D., 1620 Medical Arts Bldg., Omaha 2, Neb., **Loan.**

Demonstrates the technic of deep orbital injection for motor block of the extraocular muscles and shows the anatomy of the nerves and vessels of the orbit.

A Technique for Intravenous Pentothal Sodium with Curare in Ophthalmic Surgery
Color, silent, 25 minutes.
Year of Production: 1949; **Authors and Producers:** Murray F. McCaslin, M.D., and George J. Thomas, M.D., **Camera:** M. W. LaRue.
Distribution: Murray F. McCaslin, M.D., The Eye and Ear Hospital, Pittsburgh 13, Pa., **Loan.**

A presentation of a technic of anesthesia developed in the use of these agents in over five thousand cases.

2. Glaucoma

Cyclodialysis a Tergo
Color, silent, 6 minutes.
Year of Production: 1951; **Author and Producer:** Andrew de Roetth, Sr., M.D., **Camera:** Robert Good.
Distribution: Andrew de Roetth, Sr., M.D., 508 Old National Bank Bldg., Spokane, Wash., **Loan.**

Shows the technic of cyclodialysis a tergo and the introduction of air into the anterior chamber.

Glaucoma Surgery
Color, silent, 15 minutes.
Year of Production: 1935; **Author and Producer:** Ray K. Daily, M.D.
Distribution: Ray K. Daily, M.D., 1117 Medical Arts Bldg., Houston, Texas, **Loan.**

Illustrates the technic of glaucoma surgery.

Surgery for Chronic Glaucoma
Color, silent, 10 minutes.
Year of Production: 1948, revised 1950; **Author and Producer:** Joseph Laval, M.D., Manhattan Eye and Ear Hospital; **Camera:** Don Allen.
Distribution: Joseph Laval, M.D., 136 East 64th St., New York 21, N. Y., **Loan.**

Shows an operative technic for reducing tension in chronic glaucoma and the histology of the operative area involved. (The operation has recently been modified for use in congenital glaucoma.)

3. Decompression

Malignant Exophthalmos
Color, silent, 15 minutes.
Year of Production: 1947; **Author and Producer:** Rudolph Jaeger, M.D., Jefferson Medical College.
Distribution: C. E. J. Shannon, M.D., 9 Park St., Waterville, Maine, **Loan.**

Operation for a severe form of exophthalmos, necessitating a Naffziger on one side, and a Naffziger-Kronlein on the other. Also of interest to neurosurgeons.

4. Lid and Ptosis

Operation for Ptosis
Color, silent, 15 minutes.
Year of Production: 1938; **Author and Producer:** Ray K. Daily, M.D.
Distribution: Ray K. Daily, M.D., 1117 Medical Arts Bldg., Houston, Texas, **Loan.**

Demonstrates the technic of ptosis operations.

Operation for Senile Ectropion
Color, silent, 7 minutes.
Year of Production: 1951; **Author and Producer:** Andrew de Roetth, Sr., M.D., **Camera:** Robert Good.
Distribution: Andrew de Roetth, Sr., M.D., 508 Old National Bank Bldg., Spokane, Wash., **Loan.**

Demonstration of the operation.

Operation for Senile Ectropion. Operation for Trichiasis
Silent, 12 minutes.
Year of Production: 1939; **Author and Producer:** Ray K. Daily, M.D.
Distribution: Ray K. Daily, M.D., 1117 Medical Arts Bldg., Houston, Texas, **Loan.**

Demonstrates the technics of these operations.

Resection and Advancement of the Levator for Ptosis
Color, silent, 15 minutes.
Year of Production: 1947; **Author and Producer:** Carl C. Johnson, M.D., Massachusetts Eye and Ear Infirmary; **Camera:** Mark J. McCann, Jr.
Distribution: Carl C. Johnson, M.D., 5 Bay State Rd., Boston 15, Mass., **Loan.**

Shows external approach to levator resection and advancement.

Resection of Levator and Tarsus
Color, silent, 12 minutes.
Year of Production: 1947; **Author and Producer:** Loren P. Guy, M.D.; **Camera:** Margaret Markham.
Distribution: Loren P. Guy, M.D., 40 East 62nd St., New York 21, N. Y., **Sale:** at cost; **Loan.**

Shows technic of resecting levator and tarsus for blepharoptosis.

Total Reconstruction of Lower Lid
Color, silent, 20 minutes.
Year of Production: ? ; **Authors and Producers:** Ralph W. Danielson, M.D., and John C. Long, M.D., University of Colorado School of Medicine; **Camera:** Glen E. Mills.
Distribution: John C. Long, MD, 324 Metropolitan Bldg., Denver, Colo.; **Loan.**

Shows the removal of the lower lid with reconstruction by the Wendell Hughes technic in a case of carcinoma and one of malignant melanoma.

5. Detachment of Retina

The Operation of Scleral Resection as Used in Retinal Detachment
Color, silent, 15 minutes.
Year of Production: 1945, revised 1947; **Author:** William E. Borley, M.D.; **Producer:** Department of Medical Photography, U. S. Naval Hospital, San Diego, Calif., **Loan.**

Demonstration of a scleral resection operation for retinal detachment.

6. Lacrimal Sac

A New Cannula for Dacryocystorhinostomy
Color, sound, ? minutes.
Year of Production: 1950; **Authors:** T. S. Gerspacher, M.D., and H. D. Fowler, M.D.
Distribution: D. E. Rolf, M.D., 1017 Republic Bldg., Cleveland 15, Ohio, **Loan.**

Describes a new technic of dacryocystorhinostomy, employing a tantalum cannula. This technic simplifies and shortens the operation, and seems effective in cases of stenosis of the nasolacrimal duct and chronic dacryocystitis.

Transplantation (Implantation) of the Lacrimal Sac in Chronic Dacryocystitis
Black-and-white, silent, 10 minutes.
Year of Production: 1938?; **Author and Producer:** William H. Stokes.
Distribution: Harold Gifford, M.D., 1620 Medical Arts Bldg., Omaha 2, Neb., **Loan.**

Demonstrates the technic of lacrimal sac transplantation.

7. Cornea

Corneal Tattooing with Optical Iridectomy
Color, silent, 14 minutes.
Year of Production: 1950; **Author and Producer:** Arthur A. Knapp, M.D.
Distribution: Arthur A. Knapp, M.D., 907 Fifth Avenue, New York 21, N. Y., **Loan.**

Intended to show that tattooing in one, two or three colors may be performed at the time of iridectomy; demonstrates that the combined operation is suitable for keratoconus.

Corneal Transplant Surgery
Color, silent, 15 minutes.
Year of Production: 1950; **Author and Producer:** George M. Haik, M.D.
Distribution: George M. Haik, M.D., 812 Maison Blanche Bldg., New Orleans 16, La., **Loan.**

Shows corneal transplant with a trephine.

8. Strabismus

Strabismus Surgery
Color, silent, 15 minutes.
Year of Production: 1938; **Author and Producer:** Ray K. Daily, M.D.
Distribution: Ray K. Daily, M.D., 1117 Medical Arts Bldg., Houston, Texas, **Loan.**

Illustrates the surgery of strabismus.

Surgery of Paralytic Strabismus
Color, silent, 15 minutes.
Year of Production: 1939; **Author and Producer:** Ray K. Daily, M.D.
Distribution: Ray K. Daily, M.D., 1117 Medical Arts Bldg., Houston, Texas, **Loan.**

Illustrates the technic.

Surgery of the Inferior Oblique
Color, silent, 20 minutes.
Year of Production: 1950; **Author and Producer:** W. H. Fink, M.D.; **Camera:** Al Anderson.

Distribution: W. H. Fink, M.D., 1921 Medical Arts Bldg., Minneapolis, Minn., (no data given).

Presents some of the operations on the inferior oblique muscles.

Transplantation of the Superior and Inferior Rectus Muscles for Paralysis of the Lateral Rectus
Black-and-white, silent, 12 minutes.
Year of Production: 1951; **Sponsor:** Ophthalmological Foundation; **Authors and Producers:** Conrad Berens, M.D., and Louis J. Girard, M.D.; **Camera:** Lester Heitlinger.
Distribution: Ophthalmological Foundation, Inc., 314 East 14th St., New York 3, N. Y., **Rental:** $10.

Shows a transplantation operation for paralysis of the lateral rectus which not only utilizes the lateral halves of the vertical rectus but also shifts the nasal halves of these muscles temporally to the vertical meridian.

9. Enucleation

A New Integrated Implant for Enucleations
Color, sound, ? minutes.
Year of Production: 1949; **Authors:** T. S. Gerspacher, M.D., and H. D. Fowler, M.D.
Distribution: D. E. Rolf, M.D., 1017 Republic Bldg., Cleveland 15, Ohio, **Loan.**

Describes an integrated implant for enucleations, the operative technic, and the postoperative motility obtained.

10. Pterygium

Pterygium Operation - Arit's Technique
Color, silent, 5 minutes.
Year of Production: 1950; **Author and Producer:** George P. Landegger, M.D.; **Camera:** John B. Sullivan.
Distribution: George P. Landegger, M.D., 3875 Wilshire Blvd., Los Angeles 5, Calif., **Loan.**

Intended to popularize Arit's simple technic which, in the author's hands, gives better results than the more complicated methods.

11. Cataract

a. General

Cataract Extractions under Pentothal Sodium Anesthesia
Color, silent, 42 minutes.
Year of Production: 1950; **Author and Producer:** Clyde H. Jacobs, M.D.; **Camera:** Robert Krauss.
Distribution: Clyde H. Jacobs, M.D., Geisinger Memorial Hospital and Foss Clinic, Danville, Pa., **Loan.**

Shows the anesthesiology set-up for using pentothal sodium intravenous anesthesia in cataract extraction and depicts various types of extractions and corneoscleral sutures.

Intracapsular Cataract Extraction
Color, silent, ? minutes.
Year of Production: 1950; **Author and Producer:** Alson E. Braley, M.D.; **Camera:** Fred Kent.
Distribution: Apply to Alson E. Braley, M.D., Dept. of Ophthalmology, University Hospitals, Iowa City, Iowa.

Shows intracapsular cataract extraction; akinesis, traction sutures, keratome incision, iridotomy.

Technique of Cataract Extraction
Color, silent. 30 minutes.
Year of Production: ? ; **Author and Producer:** Ray K. Daily, M.D.
Distribution: Ray K. Daily, M.D., 1117 Medical Arts Bldg., Houston, Texas, **Loan.**

Shows the technic of cataract extraction.

Wound Closure in Cataract Surgery
Color, silent, 16 minutes.
Year of Production: ? ; **Authors and Producers:** Bennett Y. Alvis, M.D., and Edmund B. Alvis, M.D.; **Camera:** Stuart C. Mahaney.
Distribution: Bennett Y. Alvis, M.D., 308 North Sixth St., St. Louis 1, Mo., **Loan.**

Shows the closure of cataract wound employing a new type of corneoscleral sutures.

b. Intracapsular

Cataract Surgery
Color, silent, 15 minutes.
Year of Production: 1950; **Author and Producer:** George M. Haik, M.D., Louisiana State University School of Medicine.
Distribution: George M. Haik, M.D., 812 Maison Blanche Bldg., New Orleans 16, La.,

Shows intracapsular cataract extraction with conjunctival flap; cataract extraction following filtering operation; congenital cataract extraction.

Intracapsular and Extracapsular Cataract Extraction
Color, silent, ? minutes.
Year of Production: ? ; **Author and Producer:** Edward A. Roling, M.D.; **Camera:** John Mauer.
Distribution: Stritch School of Medicine, Loyola University, Chicago, Ill., **Loan.**

Shows the technic of intracapsular and extracapsular cataract extraction with sliding conjunctival flap, corneoscleral suture, etc.

Intracapsular Cataract Extraction
Black-and-white, silent, 10 minutes.
Year of Production: ? ; **Author and Producer:** Harold Gifford, M.D.
Distribution: Harold Gifford, M.D., 1620 Medical Arts Bldg., Omaha 2, Neb., **Loan.**

Shows the Verhoeff intracapsular technic, with animation of the single corneoscleral suture.

Intracapsular Cataract Extraction with Preplaced Catgut Appositional Sutures
Color, silent, 30 minutes.
Year of Production: 1950; **Author and Producer:** Wilfred E. Fry, M.D.
Distribution: Wilfred E. Fry, M.D., 1930 Chestnut St., Philadelphia 3, Pa., **Loan.**

Shows a method of intracapsular cataract extraction with preplaced appositional sutures and a peripheral iridectomy.

c. Extracapsular

Cataract Extraction, Extracapsular. Removal of the Capsule
Color, silent, 6 minutes.
Year of Production: 1951; **Author and Producer:** Andrew de Roetth, Sr., M.D.; **Camera:** Robert Good.
Distribution: Andrew de Roetth, Sr., M.D., 508 Old National Bank Bldg., Spokane, Wash., **Loan.**

Demonstrates the author's corneoscleral suture and the removal of the capsule after extracapsular extraction.

d. Erisophake

Cataract Extraction with Bell Erisophake
Color, silent, 15 minutes.
Year of Production: 1950; **Author and Producer:** I. Herbert Katz, M.D.
Distribution: Apply to I. Herbert Katz, M.D., 713 East Genesee St., Syracuse 2, N. Y.

Shows three intracapsular cataract extractions by a method which has a very low rate of ruptured capsules.

The Dynamics of Phakoerisis
Color, sound, 18 minutes.
Years of Production: 1949-50; **Sponsors:** Veterans Administration Hospital, San Francisco, and University of California Medical School, Dept. of Ophthalmology; **Author:** David O. Harrington, M.D.; **Camera:** Henry Rafael; **Animation:** Kay Hyde.
Distribution: Apply to David O. Harrington, M.D., 384 Post St., San Francisco 8, Calif., **Sale:** approx. $85; **Loan.**

Shows, by means of drawings, cadaver eye surgery and living eye surgery in slow motion and under high magnification, how the lens capsule is distorted and the zonular lamella tensed and ruptured by application of suction with a new erisophake to the anterior lens capsule, and how the capsule is ruptured by excessive suction. The intracapsular cataract extraction by forceps and external pressure is contrasted with extraction by erisophake.

The Erisophake Method of Cataract Extraction
Color, silent, 8 minutes.
Year of Production: 1949; **Author and Producer:** Everett R. Veirs, M.D.; **Camera:** Leroy Winburne.
Distribution: Everett R. Veirs, M.D., Scott & White Clinic, Temple, Texas, **Loan.**

Demonstrates cataract extraction with an erisophake, the author's method of suturing, and a new type of erisophake tip designed by the author.

A Method of Cataract Extraction with the Dimitry Erisophake
Color, silent, 12 minutes.
Year of Production: 1949; **Author and Producer:** J. H. Judd, M.D., **Camera:** Walter Craig.
Distribution: J. H. Judd, M.D., 1020 Medical Arts Bldg., Omaha, Neb., **Loan.**

Demonstrates a method of using the Dimitry erisophake in extracting cataracts intracapsularly through a round pupil.

OTORHINOLARYNGOLOGY

ANATOMY

Anatomy of Unusual Nasal Accessory Sinuses
Color, silent, 13 minutes.
Year of Production: ? ; **Author and Producer:** Fred W. Dixon, M.D.
Distribution: Fred W. Dixon, M.D., 1027 Rose Bldg., Cleveland 15, Ohio, **Loan.**

Demonstrates the anatomy of unusual nasal accessory sinuses, as seen on skulls whose sinuses have been dissected, differentially painted, and photographed on a revolving stand.

PHYSIOLOGY

The Functions of the Ear in Health and Disease
Color, sound, 31 minutes.
Years of Production: 1946-48; **Sponsor:** Doho Chemical Corp., New York, N. Y.; **Authors:** H. G. Kobrak, M.D., J. E. Hind, and R. B. Miller, M.D.; **Producer:** H. G. Kobrak, M.D.
Distribution: Medical Film Guild, 167 West 57th St., New York, N. Y., **Sale:** $250; **Loan.**

Part I demonstrates vibration of the ossicles in the normal middle ear during auditory stimulation, as seen with stroboscopic cinematography through tem-

poral bone dissection windows. Part II similarly demonstrates pathologies of the middle ear, such as otosclerosis, adhesions and nerve deafness, and closes with the complex ossicular vibrations created by band music.

Movements of the Eustachian Tube in Swallowing and Phonation
Color, silent, 15 minutes.
Year of Production: 1949; **Author and Producer:** Henry B. Perlman, M.D.; **Technical Adviser:** Dr. Schuknecht; **Camera:** Jean Crunelle.
Distribution: Henry B. Perlman, M.D., Dept. of Otolaryngology, University of Chicago Medical School, 950 East 59th St., Chicago 37, Ill., **Loan.**

Patients with large defects of the face which permit a direct view of the mouth of the eustachian tube are used to demonstrate details of tube and soft palate movements during phonation and swallowing.

PATHOLOGY

Congenital Laryngeal Stridor
Color, silent, 10 minutes.
Year of Production: 1942; **Author and Producer:** Leo Schwartz, M.D.; **Camera:** Nat Campus.
Distribution: Leo Schwartz, M.D., 111 East 56th St., New York 22, N. Y.,**Loan.**

Shows the external manifestations of obstructive laryngeal dyspnea in a 5-month-old infant and demonstrates the laryngeal mechanism which produces the phenomena of obstruction.

DIAGNOSIS

Direct Laryngoscopy
Color, silent, 15 minutes.
Year of Production: 1951; **Author and Producer:** Sam E. Roberts, M.D.
Distribution: Apply to Sam E. Roberts, M.D., 1110 Professional Bldg., Kansas City, Mo.

Demonstrates a simplified technic of direct laryngoscopy.

SURGERY

1. External Auditory Canal

Obstructions of the External Ear Canal
Color, silent, 42 minutes.
Year of Production: 1950; **Author:** Howard P. House, M.D.; **Producer:** Billy Burke.
Distribution: Los Angeles Foundation of Otology, 654 South Westlake, Los Angeles 5, Calif., **Sale:** $200; **Rental:** $20.

Shows the surgical removal by the endaural approach of bony exostoses of the external ear canal, both anterior and posterior varieties; presents the removal of a huge polyp which completely obstructs the external ear canal, with radical mastoidectomy and demonstrates the polyp's attachment at the stapes.

2. Temporal Bone

Endaural Approach to Temporal Bone Surgery
Color, silent, 80 minutes.
Year of Production: 1949; **Sponsor:** Los Angeles Foundation of Otology, Los Angeles, Calif.; **Author and Director:** Howard P. House, M.D.; **Producer:** Billy Burke.
Distribution: Los Angeles Foundation of Otology, 654 South Westlake, Los Angeles 5, Calif.,**Sale:** $400; **Rental:** $20.

Shows the endaural approach to temporal bone surgery, describing the anesthesia, the endaural incision, the simple, modified radical, and radical mastoidectomy and the fenestration operation.

Endaural Incisions
Color, silent, 14 minutes.
Year of Production: ? ; **Sponsor:** Veterans Administration; **Author and Producer:** Walter P. Work, M.D.; **Camera:** Henry Raephel.
Distribution: Veterans Administration Hospital, 43rd Ave. & Clement St., San Francisco, Calif., **Loan.**

Shows a technic for making endaural incisions.

The Fenestration Operation
Color, silent, 17 minutes.
Year of Production: 1949; **Author and Director:** Howard P. House, M.D.; **Camera:** Billy Burke.
Distribution: Los Angeles Foundation of Otology, 654 South Westlake, Los Angeles 5, Calif., **Sale:** $175; **Rental:** $20.

Shows the steps in the performance of the fenestration operation for otosclerosis.

The Fenestration Operation for Otosclerosis
Color, silent, 43 minutes.
Years of Production: 1946-47; revised 1950; **Author:** George E. Shambaugh, Jr., M.D.; **Producer:** Mervin W. LaRue; **Endoscopic Cinematography:** Paul H. Holinger, M.D.; **Camera:** Mervin W. LaRue.
Distribution: Audio-Visual Education Dept., Northwestern University Medical School, Chicago, Ill., **Sale:** $500; **Loan.**

A brief review of the physiology of the ear and of the pathology of otosclerosis, followed by animation showing the essential steps of the fenestration operation. The complete operation is demonstrated on a patient, beginning with preoperative diagnosis by hearing tests and ending with the patient three years after the operation.

Surgical Treatment of Chronic Mastoiditis
Color, silent, 42 minutes.
Year of Production: ? : **Sponsor:** St. Michael's Hospital, Toronto, Ontario: **Author:** Joseph A. Sullivan, M.D.; **Producer:** Associated Screen News, Montreal, Quebec; **Camera:** James McCormick.
Distribution: Joseph A. Sullivan, M.D., 174 St. George St., Toronto, Ontario, **Loan.**

Demonstrates a technic of temporal bone surgery for the eradication of chronically diseased middle ear structures and mastoid processes, with preservation of the two bony windows and utilization of a thin skin graft to maintain or improve hearing function.

3. Otoplasty

Correction of Prominent Ears
Color, silent, 15 minutes.
Year of Production: 1945; **Author and Producer:** Delmar F. Weaver, M.D.; **Camera:** William Loesch.
Distribution: Delmar F. Weaver, M.D., 571 Fisher Bldg., Detroit 2, Mich., **Loan.**

Shows a method for plastic surgical correction of protruding ears.

Surgical Correction of Protruding Ears
Color, silent, 29 minutes.
Year of Production: 1949; **Author and Producer:** Oscar J. Becker, M.D.
Distribution: Oscar J. Becker, M.D., 30 North Michigan Ave., Chicago, Ill., **Rental:** $3.

Demonstrates a surgical technic for correcting protruding ears.

4. Sinuses

Surgical Treatment of Chronic Frontal Sinusitis
Color, silent, 25 minutes.
Years of Production: 1948-50; **Author and Producer:** Guy L. Boyden, M.D., Oregon University Medical School; **Camera:** Charles Morris.
Distribution: Guy L. Boyden, M.D., 1735 North Wheeler Ave., Portland, Ore., **Sale:** at cost; **Loan.**

Shows a method of surgical treatment for chronic frontal sinusitis.

5. Nasopharynx

Bilateral Choanal Atresia
Color, silent, 15 minutes.
Year of Production: 1946; **Sponsor:** Medical College of South Carolina; **Author:** Richard W. Hanckel, M.D.; **Technical Adviser:** Robert P. Walton, M.D.; **Camera:** O. J. Brodie.
Distribution: Department of Medical Illustration, Medical College of South Carolina, Charleston, S. C., **Loan.**

Presents a case demonstrating nasal discharge, polydactylism and respiratory obstruction; shows the procedure for surgical correction; demonstrates the patient at intervals during postoperative recovery.

6. Rhinoplasty

The Fractured Nose (Plastic Repair for Deformity and Dislocated Septum)
Color, sound, 11 minutes.
Year of Production: 1941; **Author and Producer:** Jacques W. Maliniac, M.D.
Distribution: Jacques W. Maliniac, M.D., 11 East 68th St., New York 21, N. Y., **Loan.**

Shows a technic for repair of nasal fractures with subluxation of the septum.

The Repair of a Postoperative Nasal Defect with the Sickle-Shaped Pedicle Flap
Color, silent, 15 minutes.
Year of Production: 1948; **Author and Producer:** Delmar F. Weaver, M.D., **Camera:** William Loesch.
Distribution: Delmar F. Weaver, M.D., 571 Fisher Bldg., Detroit 2, Mich., **Loan.**

Shows the repair of a postoperative nasal defect with a sickle-shaped flap.

7. Cheiloplasty

Rhinoplastic Approach to the Cleft Lip Problem
Color, silent, 10 minutes.
Year of Production: 1949; **Authors and Producers:** Samuel Segal, Jr., M.D., and Joseph G. Gilbert, M.D.; **Animation:** Bergman Associates.
Distribution: Samuel Segal, Jr., M.D., 111 Maple St., Springfield, Mass., **Sale:** $200; **Loan.**

Shows essentials of a surgical technic of rhinoplasty, with correction of cleft lip in an adult.

8. Uranoplasty

Reconstruction of the Cleft Muscular Velopharynx and Hard Palate by Muscle Block Transfixation
Color, sound, 26 minutes.
Year of Production: 1947, revised: 1951; **Sponsor:** Fidelity Medical Supply; **Author:** James O. Beavis, M.D.; **Producer:** Film Associates, Inc.; **Technical Adviser:** Mary Quirk; **Camera:** Ray Arn.
Distribution: Fidelity Medical Supply, South Main St., Dayton, Ohio, **Sale:** $150; **Loan.**

Demonstrates on a case the surgical reconstruction of cleft palate and velopharynx with complete anatomic dissection of the muscular components of the velopharyngeal sphincter, with transfixation of the parts into their original positions. The voice and structures of the patient are demonstrated preoperatively and postoperatively, and following speech therapy.

9. Lymphoid Tissue

Technique of Adenotonsillectomy
Color, silent, 17 minutes.
Year of Production: ? : **Author and Producer:** Lewis F. Morrison, M.D.
Distribution: Lewis F. Morrison, M.D., 490 Post St., San Francisco 2, Calif., **Sale:** $75; **Loan.**

Shows details of a technic of adenotonsillectomy.

10. Larynx

Arytenoidectomy: Open Approach for Bilateral Abductor Paralysis
Color, sound, 13 minutes.
Year of Production: 1949; **Sponsor:** Otolaryngological Service, Columbia Presbyterian Medical Center; **Author:** DeGraaf Woodman, M.D.; **Producer:** Sturgis-Grant Productions, Inc.; **Camera:** Warren Sturgis.
Distribution: Sturgis-Grant Productions, Inc., 314 East 46th St., New York 17, N. Y., **Sale:** $75; **Rental:** $6.

Shows the surgical correction of paralysis of the vocal cords in a nerve injury case; the removal of one arytenoid cartilage and lateral fixation of the corresponding cord widening the glottic chink; preoperative and postoperative results, with a section on voice training of such patients.

Thyrotomy (Laryngo-Fissure) for Carcinoma of the Larynx
Color, silent, 8 minutes.
Year of Production: 1941; **Author and Producer:** Delmar F. Weaver, M.D.; **Camera:** William Loesch.
Distribution: Delmar F. Weaver, M.D., 571 Fisher Bldg., Detroit 2, Mich., **Loan.**

Total Laryngectomy
Color, silent, 36 minutes.
Year of Production: 1948; **Author:** Delmar F. Weaver, M.D., **Producer:** Davis and Geck, Inc.
Distribution: Davis and Geck, Inc., 57 Willoughby St., Brooklyn 1, N. Y., **Loan.**

Presents a case record of a method of total laryngectomy for advanced carcinoma of the larynx with laryngeal obstruction and fixation of the left vocal cord.

REHABILITATION

We Speak Again: The Rehabilitation of Laryngectomized Patients
Color, sound, 16 minutes.
Year of Production: 1949; **Sponsor:** American Cancer Society and its Massachusetts Division; **Author:** LeRoy Schall, M.D.; **Producer:** Sturgis-Grant Productions, Inc.
Distribution: Sturgis-Grant Productions, Inc., 314 East 46th St., New York 17, N. Y., **Sale:** $80; **Rental:** $7.50.

Emphasizes the specialized technic in training the patient to develop the use of esophageal speech. Knowledge and hope is given to the patient. Basic training methods are demonstrated which may be adopted in other clinics.

HYGIENE

Your Ear and Noise
Color, sound, 12 minutes.
Year of Production: 1950; **Sponsor:** Sub-Committee on Noise in Industry of the Committee on Conservation of Hearing, American Academy of Ophthalmology and Otolaryngology; **Authors:** Howard P. House, M.D., Douglas Wheeler, Ph.D.; **Camera:** Harry F. Burrell.
Distribution: Sub-Committee on Noise in Industry of the Committee on Conservation of Hearing, American Academy of Ophthalmology and Otolaryngology, 1136 West Sixth St., Los Angeles 17, Calif., **Sale:** $65.47; **Loan.**

Acquaints management and safety engineers with the problems of noise as it relates to the function of the ear; reveals methods of measuring noise in industry and describes the means of testing personnel in noisy plants; shows methods of combating the noise hazards by means of ear defenders and control of the noise both at the source and in the surrounding environment; it stresses the need for pre-employment and routine periodic audiometric tests of all employees working among hazardous noise levels.

REPORTING OF NEWLY COMPLETED MOTION PICTURES

The expanding utilization of medical motion pictures in the teaching work of the Academy members suggests that a central file of newly produced films be developed and consistently maintained. Reporting of all new films is therefore urged. The Academy will supply Film Information Forms on request.

TEACHERS' SECTION IN OTOLARYNGOLOGY

October 14, 1951

INTRODUCTION

LAWRENCE R. BOIES, M.D., *Chairman*

THE first meeting of the Teachers' Section of the Academy last year, had an attendance of approximately 75. A larger number are here this morning. (The attendance was 124.) It is significant that so many are interested in this meeting. A number of you have written to me about it.

It is intended that the Teachers' Section is to be an informal organization within the Academy to study undergraduate and graduate teaching in our specialty, to exchange ideas in meetings such as this one, and to formulate improved programs of instruction so as to advance our specialty in centers and institutions of medical education. Anyone participating in undergraduate and graduate education in our specialty is considered to be a member of this Teachers' Section by his presence here.

Last year we considered undergraduate instruction. The discussions of that meeting were published as a supplement to the TRANSACTIONS and distributed in June. Extra copies of that publication are available.

Today we will continue briefly along that same theme and then hear some views on some phases of graduate education in otolaryngology.

Several of the heads of departments among our 80 medical schools in the United States and Canada have asked for recommendations for minimum requirements for a good program for undergraduate instruction. Two years ago Dr. Schall, who was then chairman of the committee on undergraduate teaching of the American Board of Otolaryngology, presented such a program as the recommendation of his committee at an informal meeting which was the forerunner of this Teachers' Section. The official Academy Committee for this Teachers' Section approves that program. It will be presented by Dr. Gordon Hoople.

DR. GORDON HOOPLE (Syracuse): In this discussion I shall be brief. The major purpose of my brevity is to permit ample discussion of this important subject.

Undergraduate teaching of Otolaryngology is important. How much time is allotted to it and how well it is done can have significant bearing on the whole future of otolaryngology. In recent years there are many who have affirmed that Otolaryngology is a dying specialty. There is some evidence that this is so. On the dark side of the picture, there is the loss of acute suppurative surgery and the inroads which the thoracic surgeon has made in the field of bronchoesophagology. The dearth of residents and the poor quality of many applying for residency training is notable, if not alarming.

Some of us, however, believe that Otolaryngology is not dying. We admit it is a changing specialty, but we are convinced that it is gradually growing and attaining greater stature, and that more should be added to its scope. We are convinced the specialty has a great future. Those in charge of our under-

graduate teaching programs can bring much of this to pass. There are those who believe that most of the inspiration for the specialty has come from those in charge of graduate teaching. It is admitted that much can come from this area, but it should be remembered that first impressions are the important ones. The first impressions of Otolaryngology which the medical student gets are imparted to him by his first teacher, and the student's concept of the specialty is determined by the breadth of this man and the length of his vision. These impressions have much to do with the student's subsequent opinion of the specialty. To support this view I need cite but one example. Four top-flight boys are now finishing their otolaryngologic residencies. Each one is in a different training center. One is in the University of Iowa, one in Michigan, one in the Massachusetts Eye and Ear Infirmary, and one in the Manhattan Eye and Ear Hospital. They are where they are because one inspired teacher in undergraduate Otolaryngology captured their interest. First-class men will be attracted to an inspired teacher and the subject which he teaches. The second and third raters will gravitate elsewhere.

I have spoken of the teaching of Otolaryngology as it may be related to the quality of those entering the specialty. More will be said of this later in this program. What of the general practitioner? Undergraduate teaching of Otolaryngology should be primarily concerned with the preparation of the general practitioner. The preparation of the general practitioner is the first aim of all undergraduate medical teaching, and Otolaryngology has a special reason why it should support this philosophy. Lierle, in a survey in the mid-west, has shown that 25 to 30 per cent of a general practitioner's work lies in the field of Otolaryngology. Boies, in a similar survey in Minnesota, found that the general practitioners in his area estimated that 24 per cent of their practice was otolaryngologic. A smaller survey in central New York confirms the fact that 25 per cent is a reasonably accurate figure. If these estimates are correct and country-wide (and these samplings are diverse), then it is only logical that Otolaryngology should be well taught and that there should be a reasonable amount of time allotted to it. It seems to me that this is a conclusion to which all should agree.

In 1949, the recommendations of the undergraduate teaching committee of the American Board of Otolaryngology, of which Dr. Schall was chairman, were presented at a meeting of the Teachers' Section in Otolaryngology. They were:

1. The department should be autonomous.
   a. It has been found in schools where it has not been autonomous that the teaching of Otolaryngology suffers.
   b. The practice of Otolaryngology is as much medical as it is surgical.
2. General plan of undergraduate teaching.
   a. The anatomy of the ear, nose and throat should be taught by the specialty as part of the instruction in the freshman year.
   b. Requirements
      (1) Two hours of orientation, and four hours of physical diagnosis in the sophomore year.
      (2) Forty hours of didactic and clinical instruction in the junior year — arranged according to the facilities available and the discretion of the department head.
      (3) Elective courses for the senior year.

These recommendations were not official. No survey has been made since they were suggested to determine how many of the eighty departments of Oto-

laryngology in the United States and Canada have improved or changed their teaching programs.

In order to facilitate discussion, I shall stop here and ask the following questions:

1. Should this section adopt these suggested recommendations?
2. How many schools have raised their department to this level since the suggestions were made two years ago?
3. Should the heads of the departments which fall short of these requirements carry this information back to their own schools without our support?
4. Should this information, if adopted, be sent by this section to the deans of the medical schools and the chairmen of their curriculum committees?
5. Should an advisory committee be appointed
   a. To aid in establishing better standards in schools not yet committed to the program?
   b. To serve in a continuing capacity to make periodic surveys until such standards are uniform throughout the country?
6. Or, should we mind our own business?

This matter is now left in your hands for discussion and whatever action you may wish to take.

CHAIRMAN BOIES: We had a great deal of discussion last year about several of the points in these recommendations. If many of you are to speak about this again, we probably will not be able to finish this program by noon.

I can supply the answers to some of the questions Dr. Hoople has raised, and I shall ask for action on these recommendations. The latter will undoubtedly require some discussion.

In answer to Dr. Hoople's second question, I would say that we have no information on how many schools have raised their department to this level since the suggestions were made two years ago. Among those below this level, it is probable that some have considered and possibly put into effect some of the recommendations, but we have no information as to what has been done.

I do believe that there is a purpose in this group adopting these recommendations. That would "put some teeth," so to speak, into the recommendations by making them the official action of this Teachers' Section. Then those who need to can take the recommendations back to their deans. I do not believe that simply mailing anything to the dean of a medical school or the chairman of a curriculum committee will necessarily cause any action. If a program for teaching Otolaryngology is going to be improved in any school, it will undoubtedly be through the efforts of the head of a department who will, however, find his cause strengthened if he is armed with the official recommendations of this group for a minimum program. However, we will mail our recommendations to the deans of all medical schools and the chairmen of curriculum committees.

Now, would anyone like to discuss Dr. Hoople's presentation?

DR. HARRY P. SCHENCK (Philadelphia): I would like to ask a question. The bulk of that instruction is in the third year. I wonder if that were in the fourth year, would there be any objection?

CHAIRMAN BOIES: I don't think it matters a great deal whether the bulk of the instruction is in the third year or fourth year. There is argument both ways. When one trains the undergraduate in Otolaryngology in his third year, he is probably a little better equipped to do the clinical work of the fourth year. On the other hand, he has had more ex-

perience in clinical medicine by the fourth year, and if he receives his special instruction then, he is better able to give interpretations. I think we could argue these points for a long time.

DR. NORTON CANFIELD (New Haven): What response did you get from those 80 communications that you sent out? Were they also sent to the deans of the medical schools, or simply to the department heads?

CHAIRMAN BOIES: The printed transcript of last year's meeting was mailed to the heads of departments of the eighty medical schools in the United States and Canada, together with an invitation to all to participate in the activities of this Teachers' Section, offer suggestions, etc. I have received letters from several heads of departments expressing an interest in this program and offering their support.

DR. DEAN M. LIERLE (Iowa City): I don't think we are going to get to first base unless we have a definite resolution in regard to the minimum requirements for undergraduate teaching in Otolaryngology. I believe they should be sent to the deans of the medical schools, hospital superintendents and to the heads of the departments of Otolaryngology.

At the same time, I also feel that the Board requirements for examination should be sent to these same individuals.

I move the adoption of the program for undergraduate teaching as presented by Dr. Hoople as the official recommendation of this Teachers' Section.

DR. THEODORE WALSH (St. Louis): I second this motion and suggest that a copy of these recommendations be sent to the chairman of the Medical Curriculum Committee in each school. He has a great deal to do with undergraduate teaching.

The question was then put to the membership by the chairman and the motion was unanimously carried.

CHAIRMAN BOIES: Since the meeting of this Teachers' Section a year ago, I have been interested in two surveys which will be presented at this point.

A SURVEY OF RURAL GENERAL PRACTITIONERS IN MINNESOTA TO DETERMINE THE EXTENT OF THEIR OTOLARYNGOLOGIC PRACTICE

Two or three years ago Dr. Lierle surveyed the physicians of Iowa to determine what percentage of the practice of the general practitioner was concerned with otolaryngologic activities. Dr. Lierle reported that 25 per cent of the work of the general practitioner in Iowa between the months of September and June had to do with diseases of the ears, nose and throat.

Dr. Lierle suggested that a similar survey for comparison should be made in other staes. I have conducted one in Minnesota.

A questionnaire was sent out to approximately 900 physicians considered to be general practitioners in rural communities. Those listed in the Twin Cities, Duluth and Rochester were excluded. Three hundred and four replies were received. From these I discarded 48 for reasons of retirement, reply mailed from a new address in another state, or in military service, and in several instances where the physician was known to be part of a small clinic group with one member doing eye, ear, nose and throat work. This leaves 256 replies on which to base the estimates. That these 256 were from a sampling with good distribution is indicated by the accompanying chart (fig. 1).

In the questionnaire, a list of all otolaryngologic diseases was given and the general practitioner was asked to estimate the percentage of diseases of the ear, nose and throat making up his practice during the period between September and June. The replies indicated that

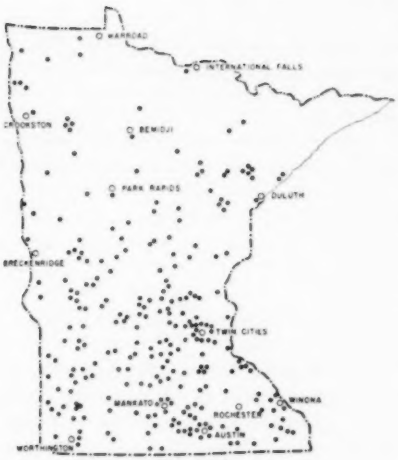

FIGURE 1.

the estimates showed an average figure of 24 per cent. It would seem reasonable to believe, therefore, that the general practitioner in the rural communities in Minnesota estimates that approximately one-fourth of his work during the season when infections are most prevalent is concerned with conditions which are part of our specialty of Otolaryngology.

A SURVEY OF UNDERGRADUATE STUDENTS TO DETERMINE WHAT THE STUDENT HAS LEARNED IN HIS CLERKSHIP

During the 1950-1951 school year, I devised a plan to make an inventory to determine just what the average student was seeing during the period of his clerkship in Otolaryngology. At the beginning of his clerkship he was handed a mimeographed list in which was included all of the diagnoses, examinations, clinic procedures and operative procedures done on out-patients and in-patients. Those items which he was expected to encounter without question were so designated. The student was asked to keep a record of his experience and turn it in at the end of his clerkship. As a result, we learned, for example, how many normal ear drums he had seen, how many abnormal drums, how many hearing tests he had observed and the type of test and hearing impairment, how many cases of chronic nasal obstruction he had observed, and how many times he had made a mirror examination of the larynx.

When I surveyed the results I was amazed at two aspects of this study. One was the large amount of elementary experience these undergraduate students were actually being exposed to, and the other one was the interest that this list stimulated in him.

This year we are giving this list to all of our undergraduates as they go through the clerkship, which consists of 45 hours of out-patient and in-patient teaching.

I have said before, and in my opinion it bears emphasis again and again, that the most fertile soil on which we can work to create an interest in our specialty and attract potential recruits of superior quality is the undergraduate student. Some of you may argue that undergraduate teaching should not include anything in the operating room, but I contend that the student who gets a supervised look in the operating room at some temporal bone surgery, a nasal or laryngeal procedure, or a chance to peek through a bronchoscope is going to be far more enlightened and experience a better chance of having his curiosity aroused than he is if his otolaryngologic experience in a clerkship is limited to observing impacted cerumen, stuffy noses, or the deafened who are having their hearing tested.

I am not going to take the time to exhibit this mimeographed list that is given to each student at the beginning of his clerkship. It covers eight pages and can be reproduced at little expense. I shall be glad to furnish any of you with a copy if you will write to me for one.

The remainder of our program is concerned with the graduate student. The question has been raised:

HAVE THE OPPORTUNITIES IN OTOLARYNGOLOGY DECREASED OR INCREASED IN THE PAST 15 YEARS?

Dr. Howard House will speak for Otology, Dr. Jerome Hilger for Rhinology and Dr. Howard McCart for Laryngology.

DR. HOWARD HOUSE (LOS ANGELES): The opportunities afforded in any specialty of medicine must be analyzed from three viewpoints, namely, the medical aspect, the surgical aspect, and the research aspect.

There has been considerable discussion regarding the evolution which has taken place in Otology during the past fifteen years. This discussion has largely followed the advent of the chemotherapeutic and antibiotic drugs.

I should like to enumerate and discuss the opportunities as they now exist in the various phases of Otology.

THE EXTERNAL EAR

Dermatologic Disorders

The otologist is best equipped to examine and treat the very common dermatologic disorders which infest the skin of the ear canal. Such diseases include eczema, inflammatory manifestations, and the fungous infections.

Otoplastic Procedures

Otoplastic procedures for correction of malformed auricles and atresias of the ear canal leave much to be desired and the otologic surgeon is assuming leadership in this field.

THE EAR DRUM

Diseases

Like diseases of the external ear canal, pathology of the ear drum is ever present and requires keen diagnostic and therapeutic judgment.

Injuries

Injuries to the ear drum are not uncommon and may result in a permanent perforation. The medical treatment, as well as their surgical repair, is as fascinating to the doctor as it is rewarding to the patient.

MIDDLE EAR AND MASTOID DISEASE

Acute Infections

Acute infections involving the middle ear should in all instances be under the close observation of the otologist. The indiscriminate use of the newer drugs in the management of acute middle ear infections too often results in hidden and unrecognized sequelae. Early myringotomy is urged in all cases which do not respond promptly to more conservative management in order that fibrosis within the middle ear does not permanently impair the hearing. Therefore, audiograms are an important phase in management of subsiding acute middle ear infections. The problem of lymphoid hyperplasia in the nasopharynx and adequate examination of this area is essential in the prevention of recurrent ear infection. Only the otologist can render this service.

Chronic Infections

The incidence of chronic suppuration will continue to be large because it is often secondary to other conditions as well as a sequela to acute middle ear infection. Close observation of these cases at periodic intervals by an otologist is essential in the interest of hearing conservation and prevention of other complications.

Chemotherapeutic and antibiotic drugs have all but eliminated surgical intervention in acute infections involving the ear and mastoid. Surgery for chronic ear suppuration will continue, however, for reasons already mentioned.

Neoplasms

Neoplasms, both benign and malignant, are not infrequently superimposed upon a chronically diseased ear. The successful management of these neoplasms requires an early diagnosis. Tumors of the ear, therefore, are the problem of the otologist from beginning to end.

Secretory Effusion

The incidence of secretory effusion is either on the increase or the condition is being more frequently diagnosed. This disease may be based on an underlying allergy, but the exact etiology still remains obscure. The immediate relief afforded these individuals by myringotomy and drainage of the middle ear fluid is most spectacular.

INNER EAR DISEASE

Tinnitus and Vertigo

The incidence of tinnitus and vertigo, either as separate or combined entities, is exceedingly large and they are frequently encountered as a major problem by the otologist. Increased knowledge of etiologic factors has revealed new fields of inquiry, both as to diagnosis and therapy. The relation of salt metabolism, allergy and vasomotor disturbances to the inner ear mechanism is well recognized.

Surgery of the labyrinth in severe labyrinthine hydrops has brought immediate relief to many patients with incapacitating vertigo of peripheral origin.

MISCELLANEOUS

Facial Nerve

The development of electronics has placed management of seventh nerve palsies on a highly scientific level. This provides a better understanding of Bell's palsy, as well as traumatic injuries to the seventh nerve. As a result, the indications for surgical decompression of the facial nerve becomes evident much earlier, resulting in a better prognosis. Technics in nerve grafting have advanced considerably in the past fifteen years.

The Temporomandibular Syndrome

Malposition and malfunctioning of the temporomandibular joint are more common causes of neuralgia and discomfort about the ear area than has been generally conceded. The successful management of this problem requires close cooperation with the dental profession.

Neuro-otology

Considerable progress has been made in the field of neuro-otology. Our present knowledge of the eighth nerve in both its peripheral and central pathways, however, leaves much to be desired. With the ever-increasing field of aviation medicine, as well as the problems of vertigo, tinnitus and hearing loss, the future of neuro-otology becomes even more evident.

HEARING LOSS

A nationwide survey has recently revealed that some 10 per cent of our population has a hearing impairment. This loss may be of the middle ear or nerve variety and may be of the congenital or acquired type. In many of these cases the hearing loss could have been prevented had these patients been under the care of an otologist from the onset of ear symptoms. Today gratifying results are being obtained in every type of hearing loss and the opportunities in this phase of Otology have multiplied many times during the past fifteen years. A few examples follow:

Middle Ear Impairment

Congenital ear atresia. Creation of a new external ear canal, together with removal of the fused incus and malleus in congenital atresia, elevates the hearing in these cases to a serviceable level, provided the hearing nerve function is within normal limits.

Artificial ear drum. The use of an artificial prosthesis, such as the Pohlman insert, may improve hearing to the serviceable level in spite of the complete absence of the ear drum and ossicles.

Perception or Nerve Impairment

Congenital nerve impairment. The Rh factor and rubella in the first trimester of pregnancy as causes of congenital nerve impairment are now well recognized. Tremendous strides have been made in the past fifteen years in rehabilitation of the nerve-impaired patient. Modern methods of speech training and the use of electronics of all types, including the hearing aid, the radio and television, have made this advancement possible. Such rehabilitation programs, both private and in clinics, must always be under the direct guidance of the otologist if maximum benefits are to be obtained.

Acoustic trauma. Acoustic traumatic hearing loss, which is commonly referred to as "boilermaker's deafness," is greatly on the increase due to exposure of more persons to high noise levels. Government, Industry and Labor look to the otologist for aid in preventing hearing loss from this cause.

Miscellaneous. Infection, vasomotor and circulatory disturbances, drugs, neoplasms and head trauma are but a few of the common causes of nerve impairment which need further clarification.

Presbycusis. The increase in life expectancy has resulted in an increase in presbycusis. This condition is universal and needs a better understanding if maximum results are to be obtained.

The private practice of audiology in conjunction with office otology offers tremendous opportunities in the care of patients with impaired hearing.

OTOSCLEROSIS

Until fifteen years ago little or nothing could be done from a medical or surgical standpoint for the patient with otosclerosis. Since that time the fenestration operation, as developed by Holmgren, Sourdille, Lempert, Shambaugh, and others, has become a practical procedure. Hundreds of fenestration operations are being performed each year which result in the restoration of practical hearing in the majority of patients. This alone is a marked tribute to the progress of otology. However, there remains much to be learned and conquered in otosclerosis.

RESEARCH

It is apparent that the opportunities for research have never been greater than at the present time. This is true whether one is interested in the medical or surgical aspects of our specialty. As our knowledge of the ear mechanism in terms of anatomy, physiology and pathology continues to increase, so will our opportunities.

CONCLUSION

As the result of the chemotherapeutic and antibiotic drugs, an evolution in otology has occurred. While these drugs have resulted in a decrease in some phases of Otology, they have at the same time made it possible for many new therapeutic technics to be evolved. These include the fenestration operation, surgery of the labyrinth, correction of congenital atresia, and many plastic procedures.

In my opinion there can be but one answer to the title of this presentation, namely, that the opportunities in otology have increased in the past fifteen years.

DR. JEROME HILGER (St. Paul): The great advances in medical knowledge of the past decades are a summation of

evolutionary, if not revolutionary, change in the component parts of general medicine. Otolaryngology had been recognized as a separate entity long before most of the so-called surgical specialties had become autonomous. As a matter of fact it became so "separatist" for a while that it, to a degree, isolated itself and its segment of the patient from the rest of medicine and surgery. For this it incurred deserved criticism.

It can be assumed that the scientific literature of a medical specialty accurately reflects the interests and accomplishments of the men in that specialty. To put modern Otolaryngology in clear light—to clarify its position in the medical world—it is good to answer three queries: From whence do we come? Where are we now? Where do we go? The special literature of thirty years ago, of fifteen years ago, and of today answers these queries in part. The direction in which we trend tomorrow also becomes apparent.

Here in tabulated form is the subject matter of Otolaryngology at these long intervals:

The distribution of subject material in the three major otolaryngic journals for the years shown are as follows:

SUBJECT	NO. OF ARTICLES		
	1920	1935	1950
Anatomy	4	10	10
Physiology	4	19	20
Pathology	2	6	10
Regional Suppuration	20	18	27
Miscellaneous Rhinology	20	35	13
Nasal and Sinus Surgery	33	39	13
Plastic	12	7	12
Allergy, vasomotor, and endocrine	3	12	13
Tumors	10	10	28

To view, specifically, the rhinologic aspects of our changing specialty:

A trend to greater interest and more frequent contributions in the basic sciences is apparent in the fields of anatomy, pathology and physiology.

Bacteriologic attitudes have been grossly modified by chemobiotic and antibiotic therapy. The nasal portal of entry relates to us much of the basic advances of bacteriology, antibiotic therapy, and the unsolved conundrum of the common cold and viral invasions.

The importance of rhinologic problems as purely local entities has faded, as is apparent in the decrease of miscellaneous rhinologic papers.

Nasal and sinus surgery from a dominant topic has faded to near oblivion. With the decrease in nasal and sinus surgery, the responsibility for maintaining a deft surgical technic has increased rather than decreased. The problems of mucocele, frontal osteoma, maxillary tumor, intranasal and nasopharyngeal new growth, polypoid degeneration and others still exist. The lessened incidence of warranted surgery only increases our teaching responsibility so that trainees can be qualified in spite of the decrease in clinical material.

Reconstructive surgery occupied a more important place in the otolaryngology of the 1920's than in the 1930's. Today the specialty reaffirms a commanding interest in the work.

The problem of architectural nasal obstruction has largely resolved itself into the proper surgical correction of the anterior third of the nose and septum. Rhinoplastic surgery has a physiologic as well as a cosmetic responsibility.

The nose and its environs, so susceptible to injury, naturally expand our reconstructive field to include maxillary surgery. The basic science of the region and the problems of surgical technic are those best known to the rhinologist.

Allergic and vasomotor viewpoints were mere voices in the otolaryngologic

wilderness of 1920. Interest erupted in the middle thirties as the futility of focal infectious and nonsuppurative concepts became more apparent. That the newer viewpoints occupy a basic position in the Otolaryngology of today becomes apparent in the broadening of the "witches brew" concept of allergy of the thirties to the present acceptance of the fundamentals of vasomotor disorder.

The nose is more exposed to environmental stress than most organs. Disturbances result which are a challenge to interpretation. Nowhere in the body are vasomotor disorders more prevalent and expressive. This implies a basic interest for the rhinologist in the rapidly progressing fields of immunology, neurovascular physiology, pharmacology, endocrinology and psychotherapeutics.

By the same token, the patient with chronic recurrent head pain has become an otolaryngologic responsibility. There is no other specialist more qualified to clarify his problems.

An aggressive interest in the neoplasms of the head and neck has developed a proper relationship to this growing field and created new responsibilities for Otolaryngology to meet. The steady increase in human longevity accentuates the problem. Refinements in surgical technic and radiation therapy must increase our salvage rate. One who deals with the primary lesions must be qualified to pursue the secondary metastases into the neck or logical treatment isn't possible.

Here, then, are the challenges in rhinology for the coming decade:

1. The maintenance of a systematic interest in the basic sciences—physiology, pathology, bacteriology and surgical anatomy — not only of intranasal structures but external nasal and adjacent structures as well.
2. The simple clarification of those extranasal and intranasal surgical procedures that have withstood the test of present critical concepts so that the art of necessary surgery will not be lost for the coming rhinologic generation.
3. The encouragement of a broad interest in the reconstructive surgery of the maxillofacial area.
4. The acquisition of available knowledge in the fields of endocrinology, neurovascular physiology and pharmacology toward the proper care of the vasomotor malfunctions of the upper respiratory tract.
5. The interpretation of vascular head and neck pain so this commonplace alien finds a refuge in Otolaryngology.
6. The application of and participation in antibiotic and immunologic advances.
7. The solution of the clinical complex wastebasketed in present thinking as the "common cold."
8. The basic preparation in anatomy, pathology and surgical technic necessary to improve our approach to the primary neoplasms of the nose and maxillae and to cope adequately with the secondary cervical involvements.

Nowhere in the entire field of medicine and surgery can a promising young man find concentrated a more engrossing combination of medical concepts and surgical technics than exists in modern Otolaryngology. The opportunity for basic contributions and clinical refinements is unlimited. Every young man who becomes a participator in rhinologic practice can readily become a contributor.

DR. HOWARD MCCART (Toronto): After World War I many men without adequate training were attracted to ear, nose and throat practice largely because of the trend in medicine to search for a

focus of infection in all systemic diseases. Medical men in general were carried away with the relationship between systemic disease and a possible focus of infection. Otolaryngology appears to have carried the brunt of the responsibility for the failures to a greater degree than necessary, because, often at the request of the internist, possible foci of infection were removed in arthritis, bronchitis, rheumatic fever, etc., and the results found wanting.

At this period Otolaryngology appears to have lost the respect of other divisions of medicine and surgery; and for some time the opportunities for expansion appeared dormant. The advent of chemotherapy and antibiotics was a later factor markedly decreasing the practice of Otolaryngology.

There is nothing we can do regarding the unsatisfactory results following the removal of foci of infection in systemic disease, nor do I think any of you here today would wish for the return of the so-called "broad field" of Otolaryngology before the advent of chemotherapy and antibiotics; but much can be achieved in the scope of our own practice in laryngology if we insist on a broad and adequate training of the men entering the specialty of Otolaryngology.

A glance at the history of laryngology reveals that it came into being as an offshoot of general medicine rather than surgery. It was not until the latter part of the eighteenth and the early part of the nineteenth century that the combined specialty of otorhinolaryngology was practiced. Today our specialty implies diseases of the ear and larynx and included diseases of the throat,pharynx, nose and nasal sinuses, trachea, bronchi and esophagus, and the Board of Otolaryngology requires that a candidate be examined in maxillofacial surgery and surgery of the neck, exclusive of the thyroid gland.

It is evident that this imposing array of diseases will not remain in our specialty permanently, as witness the loss of brain abscess and, more recently, the loss of bronchoscopy in some centers, unless we are prepared to broaden our knowledge and training.

There are many of us who have felt for some years that there is something lacking in our specialty, that it is changing, that it is becoming narrower. Perhaps it is we, the specialists, who have narrowed, and due to inadequate training, we are not capable of looking after the diseases in our sphere.

Notwithstanding these factors, I believe our field has not narrowed but is at a challenging developmental stage. We must improve on our predecessors' methods and intentions. It is my belief that this is an opportune time for otolaryngologists to reflect and lay plans rather than to wait until further inroads are made into our specialty.

These inroads are due to the weakness of some members in Otolaryngology. It could be possible that this is due to lack of interest by some of our group or the belief that there is no opportunity in their centers to do bronchoscopy, laryngology, maxillofacial surgery, etc. To those I say, grasp your opportunity before it is too late.

The present training for otolaryngologists in the United States and Canada consists of one year's general internship followed by three years' training in otolaryngology, which includes a study of the basic sciences; after which the candidate must have had a minimum of one year in practice limited to his specialty before presenting himself for examination to the American Board of Otolaryngology, or in Canada for the examination of Certification in Otolaryngology. By means of these examinations the elimination of the self-styled specialist is taking place.

In Canada, we hope in the not-too-distant future to have the Certification examination replaced by the Fellowship examination in Surgery of the Royal College of Physicians and Surgeons in the specialty of Otolaryngology. To qualify for this examination a candidate must have, in addition to the above training, two years in general surgery.

When this change takes place we would be prepared for further developments in medicine or surgery and have no fear of inroads from any department. The added training in general surgery will greatly aid otolaryngologists to become still more skillful surgeons and be competent to perform all surgery of the head and neck. At the present time the Fellowship examination is required for a teaching appointment in our medical school at the University of Toronto.

Thus, we have established certain standards for training and defined them. Some of you are of the opinion that this time is too long and unnecessary. This is not true, as surgery and its specialties, more than other divisions of medicine, demand intensive and prolonged training.

The urgent need at this time is an improvement in the graduate training of the otolaryngologist, with the establishment of higher standards. This will encourage improvement in our undergraduate teaching.

It is easy to theorize and generalize, but from a practical point of view we thought it would be interesting to present a resume of the progress that has been achieved in our Department of Otolaryngology over a period of fifteen years.

In 1935, the Department of Otolaryngology at the University of Toronto lost the control of major laryngeal work. This is best shown if we compare the work carried out in 1935 with that of 1950 in the public wards of the Toronto General Hospital, which is the largest of the University group of hospitals.

	1935	1950
Esophagoscopy including foreign bodies	27	88
Bronchoscopy including foreign bodies	54	237
Laryngoscopy	12	100
Laryngofissure	1	14
Laryngectomy	0	17
Pharyngolaryngectomy	0	2
Closure of pharyngeal fistula	0	2
Block dissection of glands of neck	0	7
Tracheotomy	3	17
Removal of thyroglossal cysts	0	7
Dilation of the larynx	0	2
Arytenoidectomy	0	4
Pharyngeal diverticulum-removal	0	3
Mixed tumors (parotid)-removal	0	4
Branchial cysts	0	3
Pharyngomaxillary abscess	0	3

In order to carry out this work we are fortunate in that the head of our Department has had a broad training and is insistent that members of his Department have a thorough training in medicine and surgery, as well as otolaryngology.

While this is a resume of the work accomplished in the years 1935 and 1950, it has really been accomplished in the short period of six years. Much of this work, such as laryngectomy and block dissection of the glands of the neck, was carried out by the Department of Surgery or the patient went to centers where this more specialized work was being performed. In many cases cancer of the larynx was treated with high voltage therapy.

While some of the major surgical operations, such as pharyngolaryngectomy, removal of thyroglossal cysts, arytenoidectomies, removal of pharyngeal diverticula and removal of mixed tumors of the parotid gland, have not been carried out in large numbers, we feel that the work has been much more stimulating than formerly.

This took courage on the part of the Department of Otolaryngology even though adequate surgical training had been taken by various members. Of even greater importance is the fact that this work was accomplished with some cooperation from the Department of Surgery so that today we feel that Otolaryngology is regaining its rightful place in the Toronto General Hospital at the University of Toronto. As time goes on and more and more men become adequately trained, we have no doubt that a larger percentage of head and neck surgery will be given over to our Department.

Numerous problems remain in laryngology for those of us who are scientifically minded, such as the relationship of Waldeyer's lymphatic ring to infection in early childhood, and the effectiveness of irradiation on lymphoid tissue in the nasopharynx. Postnasal discharge per se remains an enigma.

Much work remains to be done as to the cause of papilloma of the larynx in children and as to therapy. Cancer of the larynx and its predominance in the male are great problems for scientific study.

Laryngotracheobronchitis, its cause and proper therapy also require extensive investigation.

A new field of research has been opened up in the use of Cortisone and ACTH. These hormones control our resistance, which controls our reaction to stresses. They produce a changed reaction to infection. For example, patients with rheumatoid arthritis seldom have a cold because they are in a nonspecific hyperimmune state. They develop colds repeatedly and severely while on ACTH or Cortisone, so we realize that one of the effects of these hormones is to change the mechanism of immunity. The shrinking action of lymphoid collections in the pharynx with this hormone is important but with what benefit remains to be proven, as is also the marked alleviation of severe asthma and allergic conditions. It must be recognized that most of this investigation can only be done in teaching institutions with laboratories, by men with scientific minds and sufficient financial assistance at their disposal.

The young man of today entering Otolaryngology would appear to have an abundant opportunity in his chosen field. Leadership in medicine and our specialty is essential and urgent. So far in this country it has been allowed to develop freely and independently. If our leadership and training become weak we leave a poor heritage for those who follow, attracting youth without the necessary culture and vision. The present directors of university teaching departments of Otolaryngology must develop men who are first class surgeons, who can command the respect of their colleagues in the other departments of surgery and medicine, and the admiration of their students.In this way these men will make a great contribution towards solving the problems and expanding the scope of laryngology.

CHAIRMAN BOIES: The discussion of these three presentations will be opened by Dr. Lierle.

DR. LIERLE: The excellent papers you have just heard emphasize the opportunities that are available for the physicians who wish to specialize in otorhinolaryngology and maxillofacial surgery. The field is broad in scope; the practice is both medical and surgical. There is a challenge for those interested in research to investigate such problems as allergy, the viral diseases, malignancies, and hearing losses. A few years ago we heard from many sources that otorhinolaryngology was a dying specialty. This is far from the truth today. I have an idea that the individuals

who uttered these statements actually did not feel that way themselves but made these accusations to stir us from our lethargy and stimulate us to greater accomplishments.

About six years ago, following the discovery of the chemotherapeutic agents and the antibiotics, there was and still is a period of adjustment. If we delve into the history of Otolaryngology we find that the so-called scope has changed many times. At first there were the single specialties of otology, laryngology and rhinology as developed by the general surgeons. Later there was a combination of the three; then the field was further broadened by including ophthalmology; and later maxillofacial surgery was added. During recent years before antibiotics, the scope was again reduced because of the numerous infections of the ear and sinuses—everyone was so busy that there was little time for the rest of the field. As a result much of our work, particularly surgery of the neck and maxillofacial surgery, was done largely by other specialists. Later the field was again narrowed by the separation of ophthalmology from Otolaryngology and broadened again by increased interest in the field of allergy. There is no question that the chemotherapeutic agents and antibiotics have been important factors at this time in the desire to include again the entire field of Otolaryngology with maxillofacial surgery in the training of residents today. This really has been a boon to the specialty because it has made us aware of the fact that if we are to have entity or to survive as a specialty, we must have a definite field of work, realizing at the same time that some of our field will overlap with that of other specialties.

Is there anything wrong with Otolaryngology today? There are, of course, a number of things that could be improved but there is nothing that is so serious that it can't be remedied. I would like to discuss very briefly a few of these. First the lack of uniformity as far as the scope of training is concerned. I have had an opportunity to gain direct information concerning this by questionnaires given to candidates for Board examinations. Five years ago I don't believe you could have found similarity of the scope or field of training in any two institutions. Now about 65 per cent are including neck surgery (excluding the thyroid), maxillofacial and temporal bone surgery. The basic sciences are being taught in nearly all institutions. I am sure we are making great progress along this line. To me the biggest problem we have today is to have some uniformity as far as the field or scope of Otolaryngology is concerned in our teaching institutions. How can this be done? Heads of departments through no fault of their own, because they were trained during the period when we were too busy with infections of the ear and sinuses and tonsils to pay any attention to the rest of the field, are unable to teach the full field adequately. The training of these teachers should, in my opinion, include two years or, better, three years of general surgery, one year of internal medicine and three years of otorhinolaryngology. This can be done, because I have done it myself, and I know other institutions where it has been done.

There are many training centers today which do not have adequate material and staff to teach residents in Otolaryngology. The number of approved centers for training in our specialty at the present time has increased nearly three times since 1940. Some of these institutions obviously must be removed from the approved list.

Another thing that can be done to stimulate interest in otorhinolaryngol-

ogy is to offer facilities for research for those residents and staff members who show interest and have ability.

The present three-year period of training is not too long even for those men who go to smaller communities. It is really comparable to 18 months' training twenty years ago because three years of residence is required to see the various types of cases, and again the tempo of the average resident's work has been slowed. Now they are married, have families and in most instances leave their services at five o'clock. Twenty years ago marriage during a residency was unusual and the average resident worked every night except Sunday until ten or eleven o'clock. More time is also required for teaching the basic sciences and other subjects that have been added to the increased field and normal progress of medicine in general.

Those individuals who wish to specialize within the specialty must take extra work in addition to the basic three years training. I refer to those who may wish to devote their entire time to maxillofacial surgery, malignancies and temporal bone surgery. These individuals must necessarily, in most instances, seek large communities for their practice.

At least for the present, those going into the surgical specialties should have a special course in general surgery. This year under the present regime in general surgery would be wasted. It would be necessary for the general surgeon to arrange a special course. This would be difficult at this time.

In summary we have a wonderful specialty and the future is bright provided we have some uniformity of scope in the training of residents. To do this we need now (1) well-trained teachers with preliminary training in the principles of surgery, (2) increased interest of the medical student and the general practitioner by better teaching, and (3) stimulated research. The responsibility for these things rests with the teaching institutions. Let us go home and do something about it.

CHAIRMAN BOIES: These papers are now open for general discussion. We have two guests here from England. We would be pleased to hear from Mr. Owen and Mr. MacBeth.

MR. ROBERT D. OWEN (Cardiff, Wales): Mr. Chairman, this is quite unexpected, and I can only say how delighted I am to be here to hear your views about this very important subject. It is a great source of worry to us as well.

I was particularly interested to listen to your remarks about the questionnaire which was sent around in your State of Minnesota.

We, as you know, in England have had the old panel system going since 1911, which pertains to certification of all of those who are unable to follow their employment due to some illness or another; and when these statistics were gathered, it was found that the majority of the immediate families of the working class in particular were ill as a result of otolaryngologic infections, and our figures in England dating from 1911 varied from 2 to 24 per cent, which was very interesting from the point of view of the figures given here today.

Now, you may wonder since there is a note of anxiety here as to the future of Otolaryngology. You may ask, what are the repercussions on the specialty in England as a result of the Health Service.

You will find the statement made with regard to the general practitioner, who forms the actual hub of the profession as far as we are concerned in England, that the vast majority, I may say possibly 90 per cent, of the patients that come into the out-patient clinics of the

main hospitals are referred there by their general practitioners.

The remark that has been made now that the physicians are being overworked means this to a large extent, that the patient may go to the doctor's office complaining of something in the head and neck, and as a result of probable rush and overwork, the doctor will tell him, "I think you had better see a specialist," and a slip is sent up to the hospital for an appointment to be made for him. It may be marked "urgent," or it may not. If it is not urgent, he is put on the waiting list, and it may be that he will have to wait two, three, or four weeks before he goes to the hospital; then when these patients appear there for the first time, after waiting for three and four weeks, we find quite a large number of them coming around with nothing wrong with them. They may have had something wrong with them, say, during the first week of the complaint; but having waited, they have been cured by the waiting. The result is that there will be a certain amount of waste of time as far as we are concerned.

One other point that I think is of importance as far as the head and neck work is concerned is the fact that due to the fight for individuality among the specialties, as it were, there is a certain degree of danger in the upstartiness of some of the deep therapists with their radiation, radium, and high volume treatment; and they establish clinics of their own in the main hospitals. That is one of our headaches where there is a possibility of the general practitioner referring a head and neck cancer case to the deep therapist without having to go through the main general surgical department, and even the throat and nose department.

Now, we are fighting that, and I am certain that we will not progress from the point of view of the deep therapist having his own control of head and neck cancer surgery without having the complete cooperation of the General College.

I just want to say, sir, how much I have enjoyed listening to these papers here this morning.

MR. RONALD MACBETH (Oxford, England): It is a very great privilege for me to be here today, and I do not want to take much of your time. But I should like to say thank you, and how much I am personally in agreement with practically everything that has been said by the previous speakers.

In Great Britain, too, we have our anxieties, as Robert Owen has just said. The chest men, plastic surgeons, and the neurosurgeons are all trespassing upon our specialty, and we have a shortage of trainees. This last is partly because the young men who have been medically qualified for a year have to spend two years in the forces; when they return they are not very anxious after two years of military service and medical inactivity to begin to take up nose and throat work as a specialty.

I was not quite aware until the other day how great was the anxiety about our specialty on this side of the Atlantic. I was subjected to a very severe onslaught from the dean of a well-known medical school on this side, who told me that he thought our specialty could quite well be taken over on the one hand by head and neck surgeons, and on the other by the physicians. I put up what defense I could, not being prepared for the onslaught; and I suggested that some of our technics, diagnostic and operative, were not within the scope of a man doing only major head and neck surgery or, on the other hand, a man doing physician's work. I was referring especially to the temporal bone and nasal sinuses. I think my friend went away faintly convinced. I hope so.

I think I express the feeling that many of us have in Britain when I say that some of our troubles are due to our own fault in having let go of things that we ought to have stuck to, particularly in the surgery of the larynx and pharynx. Too many peop'e have been content to earn an easy livelihood giving office treatments and removing tonsils.

We feel that the teaching of undergraduate students should be broadly based and adequate, but not too deep; and above all, that it must be inspired. It is useless just to invite people to come and look at a few tonsillectomies or septum resections in the operating room. They will not get much inspiration from that. On the other hand, we think that our specialist trainees should have an adequate grounding in general surgery and general medicine. The English and Scottish Colleges of Surgeons insist that they should have at least one year in general surgery, as compared with the Canadian suggestion of two years, and six to twelve months in general medicine. Difficulties arise in obtaining longer training for our people in the general departments, because those departments have their own trainees to think of too. After a man has had that kind of a background together with his minimum of five years of otolaryngologic training, I suggest that he can be classified as a useful member of our specialty, and he can go into subspecialization later.

If we base our training program as broadly as we can, we should produce good, well-balanced otolaryngologists, and if we can do that we need have no fear for the future.

CHAIRMAN BOIES: Thank you, Mr. Owen and Mr. MacBeth, for your contributions.

DR. WILLIAM MCNALLY (Montreal): I beg everyone's pardon, but Dr. McCart made a little mistake in the regulations of the Canadian Royal College. The requirements for fellowship are one year rotation internship and one year in general surgery or general medicine, or a half a year of each; and three years of Otolaryngology. It is not two years in surgery. I think Dr. Ireland will bear me out on that.

DR. BENJAMIN H. SHUSTER (Philadelphia): After listening to all of the papers this morning and the statement made by Dr. Hoople, any decision made will have a far-reaching effect. Just talking, however, is not going to help us any. Making up a large list of conditions, many minor, that an Otolaryngologist can treat, and saying, "Look how much we have to do," will not help the situation nor in the end will the specialty be any bigger. If we list a lot of minor conditions and major conditions we are not permitted to do in a hospital, it will be seen how our field is restricted. Merely pinching our cheeks and pretending we are not anemic is not going to increase the importance of the specialty. There are several things that should be constructively done.

This organization, probably in association with the American Academy, ought to make such a list, stating, "The following are the things that we as Otolaryngologists are trained in, wish to practice in, are able to do," and insist upon its recognition in the hospitals.

The next step is to go to the graduate schools, and tell them, "This is the stuff that we wish taught in the graduate school so that a graduate of a graduate school, after a certain amount of training and instruction, is capable of coming out and doing these things."

And then the biggest difficulty will be when you come to your hospital and say, "I want to do maxillofacial surgery," or "I want to do neck surgery," and they will say, "No, because it is the general surgeon who does this, that, and the other thing."

You are going to have obstruction in that field from all angles. Just to cite an example or two, at the Philadelphia General Hospital we tried to get across the idea that certain neck situations—neck surgery — are also a part of otolaryngologic surgery, and whoever gets the case may do it, but we are not to be excluded. A great battle went on. The surgeons objected, "Everybody is making inroads into us. The chest surgeon is making inroads into us. The Otolaryngologists now want to take a slice out. The gynecologist does pelvic surgery. What are we going to do?" You are going to be up against those people who think you are making inroads into their field. Such arrangements ought to be made with other departments so that we should be recognized as being able to do this, that and the other surgery on a friendly basis, so that we will be recognized as having the right to do it.

Now, about the length in time of training — that has also been a very touchy subject with me. Dr. McNally has corrected Dr. McCart about the two years of surgery. It used to be one year of surgery, and there has been added three years of this, that, and another. Much time could be wasted in a hospital residency—such as clerical work and observations, to be allowed a few tonsillectomies the first year. Then a resident is allowed to do a little more next year. It is a sort of "serving time" proposition. If the hospital has material, a resident can get all he needs in one year. If there is not sufficient material or the resident is not permitted to do any work, any number of years is a useless waste of time.

I must quote a conversation I had with one of our esteemed members about this matter. I said, "I object to long years of training. I think one year of basic course and one year of residency is absolutely sufficient, provided the man goes on later and associates himself with clinics and continues to learn. Then give a man an opportunity to establish himself so that he can make a living and, at the same time, study his work. If a man wants to be a research individual, that is fine if he has special aptitude and special training." He said, "I think the several years of training is fine. My boy has taken four years of training at one of the midwestern clinics and he came back well trained. I have a very large practice and he has come in with me. He can do anything he wants." I said, "That is wonderful. How old is your boy?" He said, "Thirty-four years old. He is married and has two children." I would like to know how many of us have fathers who will keep us to the age of 34 with our families, so that they will come in with us who have lucrative practices to be taken on at once. That is ridiculous on the face of it, for the average individual we hope to attract into the field of Otolaryngology is an individual whom we ought to consider as human. In the old days, a man of 35 was usually established and earning his own living, because he was given the opportunity of earning his own living. He was practicing from the time he was 25 or 26 years old. Now, a man is gray, and a grandfather before he is allowed to say that he is a specialist. When after a few years he applies to a hospital for a position, he is told he is too old.

I think we ought not only to consider the training that the individual should get, but we also ought to consider the individual himself — that he, too, must live, and make a living, and gradually take training—without imposing a hardship on him that is almost unbearable.

Dr. Theodore Walsh (St. Louis, Mo.): I cannot agree with Ben Shuster. In the past it has been the opinion of the general physician and general surgeon that there were too many incompetent Otolaryngologists, the reason be-

ing that there was too short a training period required. I think the length of training is extremely important. I agree that a man is sometimes quite old before he can begin practicing a specialty, but with adequate training he will at least be a competent specialist. It is impossible to assimilate enough knowledge to be a competent Otolaryngologist with any less training than that which has been prescribed by the Board, as Dr. Lierle told you.

I would like to bring up another point. I think most of us are worried just now about the lack of applications for postgraduate courses. I don't know how it is in Philadelphia or in Boston, but I know that this year in St. Louis we have no graduate course in progress because we had too few applicants for it. I wonder what the reason for this is. Perhaps it is because in the past we have heard about the dying specialty and too much emphasis has been placed on the word "dying" rather than "changing." You have heard today that Otolaryngology is not a dying specialty. I am sure it isn't. I think this symposium should be publicized widely to medical students and to physicians just recently qualified, to interns and to residents. One of the difficulties seems to me to be that we spend a great deal of time training a man to be an expert in, let us say, temporal bone surgery. This man then goes off to practice in a small town where he has no opportunity to do the work he is trained for and he is left doing work of a medical nature in Otolaryngology—extremely interesting work but lacking the stimulus of some of the major surgical procedures. I can quote one particular example of this, a man who spent three years with me and was one of the best residents I ever had. He is an extremely adept surgeon and he is practicing in a small town. He gets very little temporal bone surgery to do because, as he says, "The cases are so infrequent that I don't feel that I should tackle them. I am not doing enough to keep my hand in." I think those of you who do temporal bone surgery realize that you have to be operating almost every day to keep your hand well in. What the answer to this is going to be, I don't know, unless we can get hospitals in the smaller towns with adequate facilities for these men. I think this is one of the reasons that we are not getting graduate students in the specialty of Otolaryngology these days. They don't see what they are going to do when they go to practice in small towns.

Apropos of Dr. Shuster's idea of the lack of support for fellows who are going into a specialty, we have recently been trying to change the situation by giving the residents an adequate living wage while they are in their hospital service. I think it is extremely important. The men who came in the past for residencies in the specialties had to spend two or three years in the hospital paying out money and taking in none. In some instances they get twenty-five to a hundred dollars a month, but it is hardly adequate for a man with a family to live on. I believe they should be paid an adequate wage while they are working in the hospital in their training because they do do a great deal of work for the hospital, and I believe that their pay should come from the hospital rather than from the various Departments. This matter might be taken up at some future meeting because I believe it to be extremely important.

Dr. Norton Canfield (New Haven): Dr. Lierle touched on two very important points in his discussion which I would like to emphasize. Possibly this group will be interested in recommending that some action in regard to them be taken.

The first concerns the number of approved residencies that have been started in the last few years. I think he men-

tioned that there were three times as many as in 1940. Whether or not all of these new residencies are adequate, I think, is a question. Who makes the final decision about a new residency is not quite clear. The result has been that there are now more residency positions than men to fill them, and I do not believe this trend has had the approval of the men who have been responsible for training residents. Is there anything this group can do to keep this matter in better balance? Does the American Board of Otolaryngology have any responsibility in this matter? Is it a Board function? I think it could be if the specialty members would give the Board support on its policy.

The second thing that Dr. Lierle mentioned is the attitude of the medical schools. The members of this discussion group are unified in their opinion as to the importance of the specialty in medical teaching, practice and graduate training, but in spite of our attitude, it is the medical schools who have final control over the time allotted and the facilities available for teaching. I would like to make a suggestion to this group. During the next year, could we make contact with the American Association of Medical Colleges and apprise them of our opinions? Possibly we could have a representative from their group at our next meeting to discuss this question. A direct statement of the attitude of medical schools towards the specialty would be of value. Possibly then a program of undergraduate and graduate training could be worked out for the mutual benefit of all concerned.

DR. CHARLES KINNEY (Cleveland): I would like to make one practical suggestion as to how everyone in this room could solve this problem, namely, for us as individuals to stop speaking and writing the word that has long ago outlived its usefulness, namely, the word "general surgeon."

DR. RUSSEL G. MEANS (Columbus): Being a newcomer as head of a department, I am confused over some of these high ideals.

I had a residency back in the far past when the State of Pennsylvania required two years of internship before one could take his State Board. I don't know what the National Boards now require. How long after graduation—is it one year of internship, or two years before taking the Pennsylvania Boards? Then a specialty residency of how long? I thought only three years.

Now you come along and speak of taking a year of surgical residency. What do you get in a first year of surgical residency? No more than an intern gets. They don't let them touch a knife in the first year of a surgical residency. In most hospitals one has to go into his third year or into his sixth or seventh year of senior residency before he can do any major work and before he "understands" major principles.

Is there not the possibility of rotation when you are training your men in Otolaryngology by going over to your maxillofacial group, with your plastic group, with your cancer surgery group and working in these departments when they are doing cases in our area? Then men come along who have two and three years of neurosurgery. We, too, are interested in cases involving this field. Why not interchange?

I, too, agree with Dr. Shuster that we are dragging residencies out woefully long. You demand a year of advanced medical residency after a two-year internship, a year of surgical residency and three years in Otolaryngology, then one must subspecialize when he gets to that point. One does fenestrations or something else as his major interest. One is not even qualified to do everything in the field of Otolaryngology, it seems.

I say that we need residents at Ohio State the same as, I found by writing to friends, was true in the other schools. I don't know where we are going, demanding six or eight years of postgraduate training after two years in the army. Few can afford to train that long a time. My father who was an Otolaryngologist supported me until I was 27 and could get a start That was young! They now are much older and can't afford to starve their families.

I have a son-in-law taking a medical residency and he didn't have to take all of these other "monkey-businesses" before he could become a medical resident. However, when you talk of Otolaryngology you speak of having to know everything in the world—general surgery, general medicine, etc.

I don't know what they can get the first year in any of these. Very little, I think.

I do believe that we must develop a system of rotation with allied services during an Otolaryngology residency.

DR. LIERLE: I feel that I must defend myself as far as the length of training I have advocated. In the first place, Dr. Means, I am sure you are acquainted with the requirements of the Board. They are briefly this: one year of rotating internship and three years of Otolaryngology plus a year in practice or one year's residency in surgery or medicine, or an additional year in an approved internship may be served in lieu of a year of Otolaryngology. I would say that it seems, if the Otolaryngologists were taking the usual general surgical training, that such a course should be devised to suit the needs of the specialist. I agree with Dr. Kinney that such a course should not only deal with the general principles of surgery but diagnostic and surgical procedures as well.

I can't agree with Dr. Shuster's mathematics. I don't believe that the average man completes his residency in Otolaryngology at the age of thirty-four. This may be true later if universal military training is adopted. Many individuals finish high school at seventeen and eighteen. Three years of liberal arts training, four years of medicine, a year of internship and three years training in the specialty are required. Twenty-eight or twenty-nine is the age when he is ready to practice.

DR. SHUSTER: What about the Army?

DR. LIERLE: I admit that the Army requires two years at the present time, but the individuals who are permitted to register in premedical courses have been deferred and probably in the future will be excused. We certainly hope we won't always be at war, but if we have required military training, I admit it is going to be a longer time. I agree with Dr. Walsh that hospitals have exploited our advanced residents, and something should be done about it so they may have at least a living wage and it won't be necessary for them to go deeper in debt during their residency.

CHAIRMAN BOIES: Those who believe that three years of residency training in Otolaryngology on a foundation of a good internship is too long a period of preparation would probably be the first to insist that our specialty requires as much specialized knowledge as a well-trained surgeon or internist must have. Superior preparation in these specialties requires as much and usually more than can be obtained in a three year residency.

We have to adhere to a time schedule for this program, so we will discontinue further discussion at this time and come to the last item on our program.

Another question has been raised:

WHAT SHOULD THE CLINICAL OTOLOGIST DO ABOUT AUDIOLOGY?

DR. PHILIP E. MELTZER (Boston): As director of the Winthrop Foundation for the Study of Deafness at the Massachusetts Eye and Ear Infirmary, I have had a fairly good opportunity of finding out what constitutes a complete service for the deafened. I believe I know what the set-up should be in order to render such a service. Because of my responsibility and my interest in the deafened, I have learned something of audiology.

Audiology appears to be an inclusive term embracing many divisions of science pertaining to the transmission, perception and interpretation of sound. Physiologists, physicists, psychologists, anatomists, otologists, educators and sociologists participate more or less in this new science. Because it is so new, relatively few individuals making up this composite group have been in it long enough to be classified as audiologists, in the highest sense of the word. The picture, however, is rapidly changing and needs clarification.

There apparently are different classifications of audiologists according to their education and training. Therefore, the otologist recognizes the highest category, namely the doctor of audiology, as one usually engaged in full-time activities either in a university, a medical school, or both; or, he may be engaged by a research organization or large rehabilitation center. This type of audiologist usually directs a laboratory or works in conjunction with associates in physiology, physics or psychology, all having special interest in hearing. They have been productive of many contributions most useful for diagnosis.

We also recognize the larger group of audiologists which is composed of the practical workers in the field. They are the teachers of lip reading, speech, auditory training, hearing-aid evaluation and audiometry. This capable group, holding lesser degrees in education, is one with which the otologist must become more intimate. Their special talents are indispensable.

Finally there is the audiometrist who is purely a technician qualified in audiometry and also perhaps in hearing-aid evaluation. He has a limited training but renders a valuable service to the doctor.

Now that I have made the distinction between the doctor of audiology, the clinical audiologist and the audiometrist, I should make similar distinctions as regards otologists. Relatively few otologists devote full or even half time to institutions or schools. Many give limited voluntary service to such institutions. The average otologist, therefore, is more concerned with his practice and is primarily interested in diagnosis and treatment. If he can alleviate or cure the cause of the deafness, he attempts to do so. If the hearing loss is irreversible, he may or may not be capable of following through with the proper advice to the patient. The busy otologist who practices his specialty as it should be practiced is one who performs those duties for which he is particularly fitted and refers his patient to a qualified clinical audiologist for those services which the audiologist can render best. If I appear to show partiality for the audiologist, I do so because of my experience with deafened patients who have been given the "run around" in the management of their problem. The otologist generally is up to date in medical and surgical therapy but fails to keep up with modern developments in the rehabilitation of the deafened. Herein, I believe, lies the crux of the situation as regards audiology and the clinical otologist.

Now that I have enumerated our qualifications as to where we both fit in the over-all picture of audiology, the

next question is, "How can these special talents be integrated for the greater benefit of the patient?" If the otologist fails to interest himself in anything beyond the diagnosis and treatment of deafness, he leaves himself open to critical action. By doing so, he creates opportunity for the development of spurious clinics usually directed by an intrepid entrepreneur. For this reason alone, the real hope for the proper care of the deafened lies in the establishment of rehabilitation centers supported by private or public funds.

Someone with the necessary special qualifications for this job will have to be appointed to take over these centers. Who will it be—an otologist or an audiologist? If the center is small, requiring a limited amount of time, I believe that a qualified otologist could manage it efficiently. On the other hand, rehabilitation centers with a large personnel require a full-time director educated in all phases of audiology. As this is a full-time job, there are, in my opinion, very few otologists who are prepared for it; but if one does meet the requirements, there is no reason why he should not take over. There are, however, more doctors of audiology who are more suited for the over-all direction of such a center. I see no reason to enter into any controversy over the matter. The position of director should depend entirely upon qualifications.

If we otologists leave this continually growing and articulate group of clinical audiologists to themselves, without cooperation or guidance, they will go their own way. For many years they have begged our cooperation and help. You can count on your fingers the otologists of the past who pioneered in such cooperation. Doctors Phillips, Fowler, Sr., Berry, Walker, Newhart, Hayden, Shambaugh, Sr., and Goldstein, are the names that come quickly to my mind. Fortunately there is a larger younger group who are now working intimately with audiologists. I mention this because these otologists have shown what cooperation can accomplish. If we do not cooperate, I am afraid the patient will turn elsewhere for the satisfaction he did not receive from the otologist.

If the otologist fulfills his obligations by getting together with the audiologist, the patient will certainly have received the finest kind of service and will be most appreciative. I am not relinquishing the relationship between doctor and patient. I am definitely implying, however, that if we are to hold this respected position, we must render the kind of service the patient can appreciate and be grateful for. It is really an obligation to our patient.

The question asked was, "What should the clinical otologist do about audiology?" I summarize as follows:

1. The research audiologist should be an intimate associate of the otologist and should be welcomed into our special societies. We should encourage, support and conduct researches together which will bring mutual benefits.
2. The clinical otologist must learn that his service does not end with the diagnosis. There is more to education and rehabilitation of the deafened than he knows about. He should learn how competent the clinical audiologist really is and join with him in helping the patient adjust himself to his environmental needs.

CHAIRMAN BOIES: A discussion of Dr. Meltzer's presentation will be carried over to next year's program.

Dr. Lierle has just completed a new film constructed for the elementary teaching of the undergraduate student. Some of you have seen it. I have asked him to show it in this room as soon as this meeting adjourns.

(This meeting adjourned sine die at 12 o'clock noon.)

HOME STUDY COURSES

The Home Study Courses, sponsored by the Academy, have had wide recognition as an important adjunct in the postgraduate training of ophthalmologists and otolaryngologists. They are recommended by the American Boards to their candidates and are used in conjunction with most residency training programs. Many clinicians find them valuable for a review of the basic sciences or as a guide for an organized reading program. The extent to which the Courses have been utilized is indicated by the following record of registrations since their inception in 1940:

Year	*Ophthalmology*	*Otolaryngology*	*Total*
1940-1941	301	114	415
1941-1942	238	117	355
1942-1943	163	81	244
1943-1944	126	74	200
1944-1945	175	84	259
1945-1946	290	112	402
1946-1947	353	191	544
1947-1948	436	220	656
1948-1949	375	230	605
1949-1950	317	184	501
1950-1951	244	117	361
1951-1952	301	130	431
TOTALS	3,319	1,654	4,973

Registrations should be completed before August 15 for the 1952-1953 Courses which will begin on September 1. Detailed information and application forms may be secured from Dr. William L. Benedict, Executive Secretary-Treasurer of the Academy, 100 First Avenue Building, Rochester, Minnesota.

TEACHERS' SECTION IN OPHTHALMOLOGY

October 14, 1951

Symposium: Undergraduate Instruction in Ophthalmology

The Program at the University of California Medical School

FREDERICK C. CORDES, M.D.
SAN FRANCISCO, CALIF.

As in most medical schools the time devoted to instruction in ophthalmology at the University of California Medical School during the undergraduate years is limited.

During the second year the students receive three one-hour lectures. These lectures review the anatomy of the eye and cover the examination of the eye both as part of the physical examination and in disease and trauma.

In the third year there are three didactic lectures on the infections of the eye and the trauma to the eye. These are followed by the demonstration to small sections, over a period of eight hours, of cases of infection and trauma. These demonstrations are held at the San Francisco Hospital to which the Mission Emergency Hospital is attached. This assures an ample supply of traumatic cases. During this year each student spends eight hours on the wards working up eye cases and another two hours discussing these cases with the staff or resident.

In the fourth year there are eight didactic lectures given to the class as a whole. These cover ophthalmology in a broad sense with special emphasis on ophthalmology as related to general medicine. In addition, the class is divided into small groups, and each group spends twenty hours in the clinic. Here there is a demonstration of patients augmented by projection of kodachromes of external diseases and fundus lesions. These hours are divided among the staff so that each of the men has the responsibility of covering certain phases of ophthalmology. During this time each student spends approximately six to eight hours in the wards working up surgical cases and following them to the operating room.

Thus each student spends approximately 58 hours of his time in medical school in ophthalmology. It is quite obvious that in this time it is impossible to make ophthalmologists of them, but certain things can be emphasized. The differential diagnosis of a red eye and its possibilities in relation to the individual's sight are stressed. Special emphasis is placed on the differential diagnosis of acute glaucoma and iritis.

Considerable time is spent on perimetry. The proper technic and the interpretation of the findings are discussed.

During the third and fourth years in the work in the wards of the hospitals ophthalmoscopy receives a good deal of attention. The proper use of the ophthalmoscope together with the proper adjustment of the light is stressed. An attempt is made to develop in the student a definite routine in ophthalmoscopy. In both the hospital and the clinic, whenever possible, the more common types of retinopathies are demonstrated. Where the patients are not available, fundus photographs are used in the instruction. At the San Francisco Hospital there is a wealth of ophthalmoscopic material and the students, when working up a new medical case, are encouraged to contact the eye resident

to help them with their examination of the abnormal fundus.

During the last few years one member of the staff has become interested in pediatric ophthalmology. It has been found that an hour's discussion of the eye findings in pediatric cases, including a short discussion of retrolental fibroplasia, has been of interest to the students.

While muscle anomalies are in general beyond the scope of undergraduate teaching, some discussion of the conditions resulting from motor anomalies can be made interesting and help the student at least in a general way to understand the muscle problems.

The Program at Columbia University College of Physicians and Surgeons

JOHN H. DUNNINGTON, M.D.
NEW YORK, N. Y.

At Columbia University the undergraduates receive a total of 45 hours of instruction in ophthalmology. This teaching is done largely during the third year, although in the second year one lecture on applied anatomy and preliminary instruction on the use of the ophthalmoscope are given. Twelve formal lectures to the entire class serve to introduce them to ophthalmology. These talks cover anatomy, physiology, histology, pathology, neurology and medical ophthalmology, external diseases, cataract and glaucoma. The clinical instruction is given to small groups of 8 or 10 students for 10 three-hour sessions. During this period the field of ophthalmology is covered in a systematic manner. Each subject is first outlined to the student and then illustrative cases are presented for them to examine and diagnose. At the completion of this fortnight of clinical work they are given an oral quiz, the object of which is twofold. First, it aids in assessing the type of instruction they have received and, sec-

SUMMARY OF UNDERGRADUATE INSTRUCTION IN OPHTHALMOLOGY AT THE UNIVERSITY OF CALIFORNIA

Second year			
	Three one-hour lectures on a review of the anatomy of the eye and examination of the eye as part of physical examination.		3 hours
Third year			
	Lectures on infection and trauma	3 hours	
	Demonstration of trauma and infection	8 hours	
	Working up eye cases on wards	8 hours	
	Discussion of cases with eye resident	2 hours	21 hours
Fourth year			
	Didactic lectures	8 hours	
	Work in the Clinic	20 hours	
	Working up new cases and following them to surgery	6 hours	34 hours
Total			58 hours

There is one glaring fault in the present system of teaching. The eight didactic lectures are given at the end of the fourth year so that most of the class has completed its work in the clinic before having had the didactic lectures. The curriculum committee is at present considering the possibility of having these lectures given during the third year.

One observation warrants consideration: since the undergraduate teaching in ophthalmology has been better organized and the students have been given more attention, there have been applications for the residency in ophthalmology from the top-ranking men of the class.

ond, it stimulates them to read more carefully their assignments. The clinical teaching is done by various members of the department while the quiz session is conducted by the executive officer of the department. One afternoon of the clinical period is devoted to neuro-ophthalmology, at which time they are shown illustrative cases on the wards of the Neurological Institute. The students are graded on their clinical work, the oral quiz and a written examination given at the end of the trimester. These three marks are used to determine their final grade in ophthalmology. With the limited number of hours allotted to our specialty, chief emphasis is placed upon the ophthalmological manifestations of general disease and upon the diagnosis and treatment of important ocular conditions with which the general physician may be confronted. We make no attempt to teach refraction, only outlining to them the symptoms which they may reasonably expect to be relieved by the use of glasses. In short, we endeavor to portray in an interesting manner the subject of ophthalmology not as a specialty but as a necessary part of every physician's education.

The Program at Harvard Medical School

EDWIN B. DUNPHY, M.D.
BOSTON, MASS.

At Harvard Medical School, we are fortunate in having the cooperation of other departments in teaching certain parts of ophthalmology to the medical students.

In the first year, the Department of Anatomy asks us to give two one-hour lectures in the anatomy and embryology of the eye. We also collaborate with the Department of Physiology in giving one lecture in their regular physiology course.

In the second year, we give two exercises in ocular pathology in the pathology course. In the latter half of the second year, the class is divided into three sections and each section is given an hour and a half demonstration in the use of the ophthalmoscope. In this exercise each student is taught how to identify normal structures in the fundus, using a fellow student as a patient. No attempt is made to demonstrate pathologic fundi. We also give one lecture to the whole second year class on the relation of the eye to general disease in order to orient them as they begin their course in physical diagnosis.

The bulk of the teaching comes in the third year. Here the class is divided into sections of eight men each. Each section comes to the Massachusetts Eye and Ear Infirmary every morning for two weeks. During these sessions an instructor is constantly in charge and it is his responsibility to demonstrate the common ophthalmologic conditions by using patients from the Eye Clinic. The students are indoctrinated in the use of diagnostic instruments and are instructed in ophthalmoscopy, perimetry, taking of vision, and gross examination of ocular motility. Only a very brief time is spent on tonometry and the use of the slit lamp. No attempt is made to teach the technics of refraction or retinoscopy. After the first two or three exercises, the students are assigned patients to work up and report upon. In the beginning more stress is laid upon the accurate description of what is seen than upon the correct diagnosis. The case is then discussed before the whole group from the standpoint of differential diagnosis and the relation of the particular condition to general medicine is emphasized. We do not, as a rule, allow students to observe major ocular surgery because not enough time is available. They are, however, shown the technic of removing foreign bodies from the

cornea and lids, and the incision of styes and chalazia. On the final day they take a written and practical examination.

Apart from this clinical instruction, we are allowed six didactic lectures, which are given to the third year class as a whole at the Medical School. Unfortunately, these lectures come at the very end of the year. Obviously, it is impossible to cover the whole field of ophthalmology, so only those important eye diseases met with in general practice are discussed.

In the fourth year, there is no required course but we offer an elective all-day course of one month's duration. Usually three to six students take this course each year. In the mornings, they are assigned to the Eye Clinic to observe cases with the Clinic Chief and to become more familiar with perimetry and tonometry. In the afternoons, they assist the residents in admitting patients to the house and follow up tissue biopsies and enucleated eyes in the pathology laboratory. They also make rounds with the Chief Surgeon, observe operations, and attend all clinical and pathologic conferences. Some of their time is spent in the refraction department, where the elements of refraction and retinoscopy are taught, and in the orthoptic clinic, where they have an opportunity to study disturbances of ocular motility.

Comments

EDMUND B. SPAETH, M.D.
Graduate School of Medicine
University of Pennsylvania
PHILADELPHIA, PA.

As the only member of this group connected wholly with graduate teaching, there are three comments which I can make relative to the undergraduate teaching of ophthalmology.

1. Students entering the graduate school of medicine from the various medical schools throughout the country show a remarkable lack of uniformity as to the amount of undergraduate teaching they have received in ophthalmology. Only 50 per cent of the students have a fair knowledge of the use of the ophthalmoscope, and a very small percentage of students are acquainted with the slit lamp and the perimeter. These students continue with the use of the ophthalmoscope, to a remarkable degree, throughout their internship. This may be so because such students have become interested in ophthalmology prior to or at the time of their internship. Equally significant is the fact that "knowing the ophthalmoscope they continue in its use." There is no doubt that an early acquaintanceship with the ophthalmoscope is to be desired.

2. Clinics in ophthalmology seem to be a fairly common practice for the undergraduates, but it appears that these are considered of secondary importance when compared to a set group of didactic lectures — this being the opinion of both students and teachers. I believe this to be wrong—that lectures are necessary (at least some) but that well-directed clinical sessions are much more important regardless of the student's later plans.

3. In graduate teaching of the basic sciences in ophthalmology the reverse of this is more important. Residencies and/or precepteeships have happily become so necessary that the major portion of the clinical work can be properly deferred until the start of that residency, thereby giving the student (graduate student) a much fuller knowledge of the basic sciences in his specialty.

The Program at the University of Oregon Medical School

KENNETH C. SWAN, M.D.
PORTLAND, ORE.

Teaching Objectives

1. To provide students with enough general knowledge of the common and serious diseases of the eye, adnexa, and orbit to advise patients properly concerning need for ophthalmologic care.
2. To make students aware of the importance of ocular manifestations of systemic diseases and of the relationship of ophthalmology to general medicine.
3. To qualify the student for proper emergency care of eye conditions which he might face in a general practice.
4. To ensure proper technic of examination of the eye and its adnexa in the conduct of a routine general physical examination. Emphasis is placed on the use of a hand ophthalmoscope; however, specialized instruments such as the binocular ophthalmoscope are demonstrated so that the student will be aware that there are instruments and technics which permit study of the eye in a more refined manner.
5. To acquaint students with the nature of good ophthalmologic practice so that they may give ophthalmology proper consideration as a future career and can determine who may be a good ophthalmic consultant.
6. To give the student an understanding of some of the special problems faced by patients with severe visual handicaps.

Teaching Methods

1. Lectures

Didactic lectures on ophthalmic subjects are presented throughout the four-year curriculum. Undergraduate teaching of anatomy, embryology, neurology and physiology is conducted by the basic science departments, with some guest lectures presented by members of the ophthalmology faculty. Guest lectures are also presented in courses conducted by clinical departments other than ophthalmology. These include physical diagnosis, neurology, and pediatrics. In the junior year a series of lectures is conducted by the ophthalmology department. This series covers diseases of the eye on an anatomic basis, diseases of the choroid, lens, etc. In the senior year an additional course of eleven lectures is given with emphasis on the relationship of ophthalmology to general medicine. No attempt is made to teach surgery, but by the use of motion pictures, the students are given an understanding of the nature of eye surgery. Nearly five hundred slides are used to illustrate the undergraduate lectures.

2. Laboratory Teaching

Laboratory hours in the basic sciences related to ophthalmology are given in four departments. Decision as to the material taught is made by the basic science departments, but these departments have sought advice from the ophthalmology staff so that their teaching can serve as a basis for later clinical work. Interdepartmental conferences have been particularly important in teaching the students consistent terminology. During the course of the senior year each student participates in one clinical-pathological conference conducted in the John E. Weeks Memorial Laboratory in Ophthalmology.

3. Demonstrations

Demonstrations are made for groups of three to five senior students. In addition to showing eye patients and teaching the use of the hand ophthalmoscope, the special-

ized instruments used in ophthalmology are demonstrated to give the student an understanding of how these instruments might be used to help him in consultations concerning certain general diseases. No attempt is made to teach the student the technic of using this equipment. Also demonstrated are technics of applying bandages, removing conjunctival foreign bodies, and other such simple emergency procedures as might be required in a general practice.

4. Clinics

Each senior student spends seven two-hour periods working in the eye clinic. There are few students who are qualified to conduct and record an adequate history and examine patients with serious eye pathology; however, all students are given some responsibility for care of patients by assisting the resident staff with their routine clinic work. Usually two students work with one resident.

5. Hospital Ward Walks

Each senior student spends seven two-hour periods studying fundi of patients in the medical and neurologic services. Ninety minutes are devoted to examination of patients and thirty minutes to oral quizzes and discussions by members of the ophthalmology faculty. Five to seven students are assigned to one instructor. An assistant resident locates the patients and dilates their pupils prior to the class.

6. Observation of Surgery

Observation of eye operations by students is permitted but not required. Short motion pictures of strabismus, glaucoma, and cataract surgery are presented during the lecture period to provide the students with some understanding of the nature of ophthalmic surgery.

7. Elective Classes

An opportunity is provided for senior students with special interest in ophthalmology to take extra work in the eye clinic, participate in staff conferences and observe surgery. This elective work, with or without credit, provides an opportunity for students to evaluate ophthalmology as a possible career, as well as an opportunity for the faculty to recognize and encourage students who demonstrate outstanding qualifications for this specialty.

8. Student Assistantships

For many years our faculty has selected from the freshman and sophomore classes a few outstanding students to be appointed to part-time positions in the basic science departments. These students, entitled Assistants, have participated in the teaching and research program as members of departmental staffs. Most of these students have spent five years completing the usual four-year curriculum but have been rewarded by additional educational opportunities and the salaries which these positions have made available to them. Many have been encouraged to continue careers in medical education. Most of the others have been outstanding in private practice. Our faculty feels that these student assistantships offer the exceptional student an opportunity to broaden his education beyond that of the regular medical school curriculum and thereby to become a better doctor. In 1946 a student research Assistantship in ophthalmology was made possible by a donation of $5,000 from Dr. Frank B. Ralston, a practicing ophthalmologist. This fund, which has been doubled by donations from other physicians, earns nearly four hundred dollars yearly. Each year, this sum is paid as an honorarium to a medical student selected to assist in the laboratory phases of re-

search in glaucoma. The Frank B. Ralston Assistantship has been made such an outstanding phase of our undergraduate program that additional funds are being sought to establish two additional student assistantships, one in ophthalmologic pathology and another in ophthalmologic bacteriology and virology.

The Program at Northwestern University Medical School

DERRICK VAIL, M.D.
CHICAGO, ILL.

One of the most instructive of committees on which I have had the privilege of serving was that of this Academy on undergraduate teaching. The report of this committee will be found in the TRANSACTIONS, November-December, 1947, page 134. Perhaps you have forgotten about it, if so I urge all of you to look it up, for the recommendations it contains have been of great value to me personally at Northwestern and we have tried to follow them in our courses to the undergraduate. I can attest to the soundness of the plan, I can also say frankly that there have been some difficulties encountered in trying to approach the ideal. But the underlying philosophy has always been that of trying to instill an interest in ophthalmology and its practical use in general practice. Up to the present we have failed for a number of reasons in getting across what is to my mind the most important part of our teaching, and this is ophthalmoscopy. This year, however, the schedule has been changed, thanks to a reorganization of the Clinics, etc., at Northwestern and I am confident that our department will be better able to put across the teaching in a more stimulating fashion.

The 135 Juniors as a class are given 10 one-hour lectures during the winter quarter. Emphasis in these lectures is placed on anatomy and physiology, conjunctivitis, uveitis, glaucoma, ophthalmoscopy and neuro-ophthalmology. This lecture course is unchanged from that of the past. In addition, the entire class will meet at the Cook County Hospital for one hour a week during the fall quarter (approximately 10 to 11 periods). Here patients will be shown, lecture points emphasized, audiovisual methods employed, and quizzes freely utilized.

The new and, to me, the most exciting opportunity is that afforded by an hour of section (7 men) work on the wards each week for six to eight periods. Five hospitals in Chicago affiliated with Northwestern will be used concurrently for this work, under the various faculty members of the department in their respective hospitals. Heavy emphasis will be placed on ophthalmoscopy. Each Junior student is required to own an ophthalmoscope and will be taught its instrumentation. Every effort will be made to gather suitable clinical material together on the wards. This clinical bedside teaching comes after the didactic lectures and will be utilized to the best advantage in tying in the didactic information, particularly ophthalmoscopy. It is hoped, therefore, that every student will have learned enough practical ophthalmoscopy to be stimulated in pursuing this interest for the rest of his professional life.

In the Senior year, sections of three students daily for two weeks are assigned to the out-patient department for three to four hours a day, throughout the year. I am looking forward to the stimulus to our faculty members by the opportunity thus afforded to do better teaching. The students see new patients and are responsible for their work-up, with the desirable point of view that the student is the patient's physician and the attending faculty member is the consultant, rather than the student being

merely an observer — and how tiring that is.

I believe that this plan for undergraduate teaching for the first time at Northwestern more nearly approaches the ideals set forth in the committee's report and will stir up the enthusiasm and teaching vigor of our department to an exciting pitch.

The Program at Washington University School of Medicine

LAWRENCE T. POST, M.D.
ST. LOUIS, MO.

The program at Washington University is as shown in the following outline:

Freshman Year

Two one-hour lectures in the course on neurophysiology of the eye and visual tracts of the eye.

Three hours of experiments including optics, color vision, etc.

One-hour lecture and two hours of laboratory on the anatomy and histopathology of the eye.

Sophomore Year

Three hours of demonstration and lectures in physical diagnosis. The sessions cover the construction, principles, and use of the ophthalmoscope, the normal fundus, and common fundus pathology.

One-hour lecture and two hours of laboratory on the pathology of the eye.

Junior Year

A total of twelve hours didactic lectures. These lectures cover the most important phases of clinical ophthalmology at the undergraduate level.

Senior Year

A total of twenty-two hours of clinical instruction and demonstration in the out-patient clinic. Small groups of four or five students spend five afternoons for two weeks in the ophthalmology out-patient clinic, where they are given instruction in diagnostic technics and methods. They work up the patients for demonstration and one two-hour period is spent observing surgery in the operating room.

Except in the fourth year, the lectures and laboratory work are given to the class as a whole. The textbooks recommended are "Gifford's Textbook of Ophthalmology" by Francis H. Adler and May's "Diseases of the Eye."

In the fourth year a full-time elective for six weeks is offered. Not more than one student at a time participates in this work. He serves as a clinical assistant in the Department and takes lectures and attends demonstrations given to the graduate students in ophthalmology.

All courses are compulsory except the elective just described.

We believe that this presents an adequate program for undergraduate students for whom we think that the important elements to stress are the relationships of ophthalmology and general medicine and the giving of a good understanding of the commoner diseases of the eye.

SUMMARY OF UNDERGRADUATE INSTRUCTION IN OPHTHALMOLOGY AT WASHINGTON UNIVERSITY

	NO. OF HOURS	YEAR
Lectures and demonstrations	3	Freshman
	4	Sophomore
	12	Junior
Laboratory	5	Freshman
	2	Sophomore
Out-patient clinic	22	Senior

INDUSTRIAL OPHTHALMOLOGY

INTRODUCTORY STATEMENT

If anyone in the field of Industrial Medicine has thought through the over-all picture of industrial medicine in a clear-cut basically sound and simple form, it is L. Holland Whitney, M.D., who first appeared on the industrial medical scene as Medical Director of the Sperry Gyroscope Company during the war. Doctor Whitney did a magnificent job and one which has become a pattern to many industries. He is now with the New Jersey Telephone Company and again doing a fine job. It has always been a problem to present to specialists (in this instance, ophthalmologists) the over-all industrial medical picture in such a way as to interest them and to have that picture indicate the way an eye program ties into it. Doctor Whitney has done just this.

It is always important to get viewpoints of ophthalmologists working with various industries, especially those ophthalmologists who have varying attitudes toward industrial eye programs and eye work. It is a pleasure to include in this issue Doctor Vail's paper, "Conditions Affecting Visual Efficiency in the Railroad Industry."

HEDWIG S. KUHN, *M.D.*
Secretary, Joint Committee
Industrial Ophthalmology

WHAT A VISUAL PROGRAM MEANS TO EMPLOYEE, MANAGEMENT AND THE COMMUNITY

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BY INVITATION

VISUAL performance has a direct influence on the health, the safety and the efficiency of the worker. It is stated that 80 per cent of all impressions come to the worker through his eyes. They are the only tools which every individual must use, regardless of his specific job undertaking. It is appropriate, therefore, that industry should give consideration to the proper utilization and conservation of this important sense of seeing. A vision program designed to meet the needs of a particular industrial group results in benefits to the employees, the management and the community.

Modern management recognizes manpower as its most important asset. Industrial health programs have been developed to serve employees and thereby assist management in the most effective use of that asset. There is now available a considerable amount of evidence to indicate that careful consideration of vision is an important factor in a well-balanced program of industrial health.

Visual performance lends itself admirably to an improved utilization of manpower because it can be measured, and in a large percentage of those with inadequate vision, something can be done about it. Furthermore, a study of the various jobs in any industrial organization reveals that the visual requirements for efficient performance can be defined. Thus it is possible to fit round pegs into round holes, and in contrast to many other human characteristics, where the peg tends to be square, in many instances it can be reshaped.

Medicine found its way into industry with the advent of the compensation laws in the various states, beginning about 1910. Employers soon found that the expense of caring for the injured employee could be reduced materially by early treatment. Not only did this reduce the direct expense of medical and hospital care, but it reduced the amount of time lost from production. There has been a very steady growth and development of this early concept until the

Medical Director, New Jersey Telephone Co.
Presented at Conference on Occupational Vision, Rutgers University, Nov. 1, 1951.

present time. The emphasis has now shifted quite properly to accident prevention and health maintenance. Both World War I and World War II proved to be catalysts to this advance. The magnitude and urgency of World War II, with its unprecedented demands on production, resulted in the absolute necessity for keeping as many employees on the job for the maximum amount of time, and in the best possible state of efficiency. *Machine maintenance* in industry has long been considered a vital function in the continued success of the operation. It is encouraging to note increased emphasis on *human maintenance*, and the gradual recognition that it is at least equally important in the productive welfare of the organization.

Broadly stated, the objective of an industrial health program is to keep employees on the job in the best state of health and efficiency possible. There is no conflict here between employee and employer, because it is invariably true that what is best for the employee is also in the best interest of the employer. It is not my assigned purpose to describe all of the various technics by which this objective is attained. However, among others, preplacement examinations, periodic health examinations, and accident prevention occupy a place of high priority. Now let us consider how vision is related to each of these three technics.

In the early years of industrial medicine a preplacement examination was accomplished in a few brief minutes. It usually placed emphasis on the condition of the heart and the presence or absence of hernia. The more modern concept of this procedure calls for an appraisal of the physical and mental capacities of the applicant and matches this information against the analysis of job demands. This refinement resulted largely from experience during the manpower shortage of the last war, when it was demonstrated that many individuals who were formerly considered unemployable are actually capable of first rate performance provided they are given jobs which are suited to their individual abilities. The medical recommendation regarding placement is then integrated with the judgment and final decision of the personnel office and the operating department.

Although the Snellen test of distance vision has proved useful for medical-legal purposes and gives fairly accurate information regarding visual acuity at distance, it falls short of the goal for at least two reasons: (1) it provides data on a single visual function only, and (2) it is usually applied to a visual standard for the proposed job that is arrived at arbitrarily. Since an analysis of visual skills can be obtained quickly and easily by a visual performance testing instrument and this information can be matched against *objectively* determined visual requirements of the job, careful attention to this phase of the examination proves to be a significant refinement in placement technic.

Periodic health examinations are also a basic tool in the human conservation effort. Medical authorities have long recommended periodic health appraisal by the family physician to a public that is apparently not yet fully aware of the benefits resulting therefrom. Regular dental check-up is more readily accepted, perhaps because of the dread of a toothache or because this important phase of health conservation lends itself more readily to commercial advertising. However, periodic health examinations in industry on a voluntary basis help to overcome the inertia or resistance of a considerable segment of our population. By this means it is often possible to detect early deviations from normal which have not yet resulted in recognizable subjective symptoms. In such cases early treatment by the private physician frequently prevents needless pain and

suffering and avoids unnecessary economic loss to both the employee and the employer. The objective is to assist the employee in maintaining those capacities and skills possessed when he entered the industry. Periodic appraisal of the visual performance is an important feature of such examinations, and this identifies changes in vision which are known to occur gradually with advancing age.

The irrevocable waste in human resources by preventable injuries is a national tragedy. Statistics which develop this point are readily available and this subject will receive more attention by speakers who follow. Industry is concerned about accidents because of their cost in human resources and insurance dollars. The National Society for the Prevention of Blindness and the New Jersey State Commission for the Blind have been carrying on an effective program for many years to eliminate, or at least reduce, those accidents which destroy or impair vision. In the State of New Jersey today there are about 6,000 to 7,000 blind people. At least 60 to 70 per cent of this blindness might have been prevented. Employees engaged in any type of work which exposes them to eye hazards require eye protection, and those who have been responsible for enforcing the use of safety glasses know that eye protection is not accepted by those who need corrective lenses unless that correction is incorporated in the safety glasses. A test of visual performance frequently identifies employees who need a correction, a need that had not been previously realized. Properly prescribed and fitted safety glasses will almost entirely eliminate eye accidents. But also as a by-product of this activity we find that better vision for the job reduces the frequency of all kinds of accidents.

The premise is, therefore, that visual performance is an important human asset which deserves careful consideration in at least three phases of industrial health activity; namely, job placement, health maintenance, and accident prevention. By such consideration everyone profits, the employee, the management, and the community. My experience in two large industries during the past eight years would seem to support this contention.

The first of these companies is engaged in the development and production of precision instruments. During the war it was necessary to expand rapidly, and large numbers of people, mostly women without previous industrial experience, were hired. Our attention was first directed to the problem of visual performance because it was becoming increasingly difficult to teach inexperienced employees one important calibration job. An average of one out of every four girls, given three weeks of intensive training, was unable to produce satisfactorily either in quantity or quality. All employees so assigned had passed a preplacement medical examination, including a Snellen test for visual acuity. Interviews in employment and testing for intelligence, manual dexterity, etc., indicated that the applicants assigned had all received satisfactory scores. Further investigation of the job itself showed that satisfactory performance required special visual skill at near point. A visual job profile was developed by studying all operators on the job, and only those applicants whose vision met this standard were assigned to this job. By this refinement of placement, failures were reduced from 25 per cent to less than 5 per cent. The principles illustrated by this experience were found to be applicable to many other jobs in this industry, where vision is a critical factor in nearly all departments.

More recently in my present industrial position, records have been kept on 1,500 applicants who have been examined during the past five months. A

great majority of these applicants are 25 years of age or less. Ninety per cent of them were found to meet the visual standards for the proposed job, but 10 per cent did not and were advised to have their eyes examined by an eye specialist of their own choosing and return for re-examination. About 65 per cent of the group so advised did return after complete eye examination, and of these, 85 per cent were then found to meet the job profile.

Despite the fact that this country enjoys the highest health standards in the world today, there are many who, through ignorance or neglect, do not receive adequate eye care. It has been estimated that 30 per cent of our population would benefit from eye care that they have not sought. Selective service statistics give credence to this estimate. Obviously, the exact figure will vary with the economic and social level of the community, just as the visual demands of one industry will vary from another.

Because of the high incidence of visual inadequacy found among older employees at the time of periodic examination in the first mentioned industry, 300 machine operators who were 38 years of age or over were given complete eye examination. Nearly all of these men had been associated with this company for many years and were highly skilled. Their work consisted of "machining parts" to a tolerance of 1/10,000 of an inch. The older age group was selected deliberately in order to study those employees who were in the so-called presbyopic age group. The results seemed significant. Of this group 68 had never had previous eye examination. Fifty-two of these 68, or 76 per cent, needed correction for the job they were doing. Sixteen, or 24 per cent, did not require eye care.

Of the 232 who had been previously examined, all were wearing glasses. Sixty-four per cent, or 149, required a change in their prescription. Thirty-six per cent were found to be adequately corrected. The time between professional eye examinations among the 232 who were wearing glasses ranged from one month to 27 years. The average time between examinations was two and one-half years, even though these men were doing work which required critical vision. Ten per cent of the group had not been examined in five years.

Preliminary studies made on several hundred employees in the various departments of the Telephone Company to establish visual standards indicated clearly that an extension of this service to all on-force employees would be desirable. Accordingly, the vision screening test was made available on a voluntary basis to the entire company. Approximately 15,000 employees participated. Testing was done by trained operators at the work location, and all results were forwarded to the medical department for interpretation. The established job standard did not require visual performance as high as might be true in many industries. Despite this fact, when the results were analyzed, about 10 per cent or 1,500 employees were found to need a complete eye examination. They were so advised by letter from the medical director, and it was suggested that they seek an eye examination by a doctor of their own choice. Final tabulation of follow-up studies is not yet complete, but preliminary observations indicate that a large majority of this referred group was in need of refraction or other definite eye care. In no case did the result of the screening test or subsequent eye examination have any influence on the employee's job assignment or security.

From these two experiences related above it seems safe to generalize that a considerable segment of our industrial population is in need of eye care, a need which is not recognized until some defi-

nite symptoms develop that the average worker can associate with defective vision. Periodic examination of the employee's vision will identify many who need eye care.

Numerous studies have been made which demonstrate the relationship between visual efficiency and safety, both as regards accidents generally and accidents to the eyes. In the manufacturing company previously referred to, as a result of rapid expansion with unskilled workers who had no previous factory experience, the accident frequency and severity went up sharply. Eighteen months after the institution of an eye protection program as part of an over-all safety effort, the accident frequency was reduced by 54 per cent and the severity rate by 29 per cent. The loss ratio for compensation insurance was reduced from 84 to 51 per cent. Over a period of two years, 40 pairs of safety glasses were on exhibit that had prevented a serious eye injury. The incidence of "eye cases" treated in the plant hospital for foreign bodies, minor lacerations, splashes, etc., was reduced by more than 60 per cent. Although it is impossible to estimate the effect of improved visual efficiency on the over-all accident rate, it was surely one of the major factors in the improved accident record mentioned above.

In the plant department of the Telephone Company, a study was made to determine if there was any direct relation between visual efficiency and accidents. Seventy-nine installers and repairmen who had had one or more "personal injury" or motor vehicle accidents during the last two years were watched with 79 other men of the same age and experience, and in the same craft and district, who had had no accidents. The former were put in the "accident group" and the latter in the "accident-free group." When the plant craft vision standard was applied to these two groups, it was learned that substantially more men in the "accident group" failed one or more tests in the vision standard than did those in the "accident-free group." Three times as many men in the "accident group" failed two or more tests in the vision standard than did the men in the "accident-free group." Quite similar findings have been reported from various other industries.

The experiences and observations related would seem to substantiate our original premise that visual performance is an important human asset which deserves consideration in job placement, health maintenance and accident prevention. Many articles recording similar data have been published during the past five years in personnel, trade, scientific and medical journals. The urgency of the need for a vision program in a given industry will vary with the nature of the work and the average age and economic status of the employee group. Uniformly significant results, however, can be obtained in any industry if the health, the safety and the performance of the employees are used as instruments of measure.

Employees, management and the community eye specialists are all concerned initially as to how such a vision program in industry will affect them. The employees are anxious to know what will happen to them if their vision is found to be substandard. In my experience, not one employee has ever lost his job or suffered a reduction in pay as a result of this activity. The employee group and the labor representatives deserve and should have a complete explanation of the motives, methods, and anticipated results. It is soon apparent to the employees that visual efficiency has a direct bearing on the general level of health, reduction in fatigue resulting from eye strain, increased efficiency and greater safety.

Likewise, management must be sold on the practicability of a vision program in the industry. Unless the employees are convinced, there will be no participants, but without the backing of management, there are no funds. Industrial management is made up of practical businessmen who are not primarily interested in a fancy accumulation of medical diagnoses or statistics. But when the effects of the vision program are explained on the basis of human maintenance, it is not too difficult to establish the effect on training time, labor turnover, sickness and absence costs, reduced spoilage and accident expense.

Improved vision must also surely have an effect on the community in general. We can only speculate on what better vision generally might do to reduce the appalling number of accidents on our highways. It might even make possible a more equitable distribution of chairs around the television set. Certainly the eye specialists in the community are in a position to benefit from an industrial program, not only by the actual cases sent out for eye examination, sometimes before it is too late, but also by the resulting health education in the families of employees who are benefitted.

And so, as one of our famous orchestra leaders would say, "Everybody's happy." But before this offering ends on a note of over-optimism, a few words of caution are in order. First, if a vision screening instrument is to be used in an industry, employees should have carefully explained to them that the information obtained is only a test of how well they see. It is not meant to take the place of regular complete eye examinations, nor does anyone pretend that a screening instrument will detect a pathologic condition in the eyes. And certainly it doesn't tell definitely whether or not a person needs glasses. It will, if properly operated, determine with fairly high accuracy the visual skills or performance of the one being tested. If the instrument is accepted and used in the way it was intended by the manufacturers, it is a useful tool, but not a black box which contains magic.

Admittedly, there are many factors which influence the health level of any business. No claim is made that a vision program will cure, by itself, all the maladies of absenteeism, labor turnover, insurance costs or the more complicated ailments relating to labor relations. However, a well conceived vision program properly integrated into the personnel, safety and medical activities of any industry has sufficient influence to validate the effort.

CONDITIONS AFFECTING VISUAL EFFICIENCY IN THE RAILROAD INDUSTRY

DERRICK VAIL, M.D.
CHICAGO, ILL.

THE railroad business represents a cross section of all industry. Its materials for the most part are massive and resistant, and its manpower is anatomic. From a background of experience with ocular conditions and injuries suffered by the operating personnel of railroads, I shall discuss some of the factors and conditions affecting their visual efficiency.

Active railroading is hazardous. Its job is that of moving people and things in huge machines on steel rails in sunshine and glare, twilight and darkness, fog, mist, sleet, rain, snow and wind. Movement and speed are its commodities, and the greater the speed, the greater the demand on its men and materials. It is small wonder, then, that injuries to all parts of the anatomy of its active

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workers are daily occurrences. The wonder is that they are not more frequent and more mutilating and fatal than they are. This is a tribute to the extensive and efficient safety measures adopted by all railroads for the prevention of accidents, to the skill and training of the workers, and to the skill and efficiency of the medical and allied professions having to do with treatment of these injuries.

The visual requirements of active railroading vary enormously, depending on the individual job. Those whose work it is to direct the propulsion of the massive trains of today must have the highest quality of visual efficiency, whereas those who herd material, animals, and people into and out of trains may safely have a lower quality of visual efficiency. If an army moves on its stomach, a railroad may be said to move on its eyes, even if the miracle of its movement is guided by steel rails and protected by rights of way.

The visual efficiency of an individual depends first upon visibility, then upon the health of his eyes, his distance and near visual acuity, the extent of his fields of vision, the range and coordination of his ocular motility which includes his ability to fuse the images of his two eyes, his color vision, and his bodily reaction time to the stimulus of his sight. Thus a condition that affects any one of these factors reduces by so much one's visual efficiency on his job.

Of the factors that enter into the composite whole known as visual efficiency, the first is the visibility. To be seen, a body or object must be luminous. The factors that influence railroad visibility have already been touched upon. The human eye possesses two qualities of vision. These are photopic or vision in light, and scotopic or vision in semidarkness. The first is the function of the cones of the retina. These are accumulated in the macular region and constitute central vision. The latter is the function of the rods, accumulated in the periphery of the retina, and constituting peripheral vision. Central vision is inefficient in darkness, peripheral vision is less efficient in light. The efficiency of night vision decreases with age, illness, malnutrition, and long exposure to glare. This brings up the matter of the health of the eye that is to perceive the luminous objects.

This second factor influencing the visual efficiency of the railroad worker embraces the entire field of ophthalmology, for the worker is a human being subject to all of the ills of that gracious state. The general health of the worker is, therefore, our first consideration. Since the eye is a part of the body, diseases of the latter often affect it. There is no need here to discuss these conditions except to give examples. It is well known, for instance, that diabetes and vascular hypertension often affect the retina by hemorrhages, ischemia, exudates and edema. These lesions may be peripheral in the retina, or affect the central vision, or both. In addition, early diabetes often produces a refractive error, generally myopic, as an early symptom.

The third factor, distance and near visual acuity, is one of the most obvious. Thanks to the pre-employment and subsequent yearly examinations that are universally required, most railroad employees who need glasses are uncovered, and on the whole are satisfactorily refracted. At these times, too, diseased conditions within the eye are discovered and either corrected if possible, or, if not, other disposition of the employee is made.

The distance and near vision of an individual are affected by age changes that are almost physiological. Lens sclerosis and opacification of the lens nucleus are frequent concomitants of age and are sometimes found to be pres-

ent in the early fifties. If these changes are significant, vision of distance can be increasingly affected. Presbyopia is a physiologic change, too, and unless corrected interferes with quick and accurate reading of dials, figures, etc.

Here the important matter of seniority enters the argument. Since many of the engineers who arrive at sufficient seniority of service to be assigned the crack locomotives are past 58 years of age, it seems likely that their lenses may be sclerosed, particularly the nucleus of the lens. At this age, too, sclerosis of the retinal vessels can be found, and, if associated with hypertension, the retina and optic nerves can be affected. Cerebral changes as well are often associated with the ocular changes. They may affect memory and judgment. Work experience and know-how compensate in some measure for the physical changes found within the eye, but more attention should be paid to the ocular changes of age, particularly in the engineers, than is now customary.

The Armed Forces are now engaged in many important studies revealing the effect of speed on visual efficiency, and thus we are finding out more about this factor. A recent article by Colonel Victor A. Byrnes, of the U. S. Air Force, on "Visual Problems of Supersonic Speeds" discusses many of the conditions affecting vision at high speeds, a number of which play no part in even the high speeds attained at times by trains, but do suggest that a study of these factors applied to land speeds would be instructive. These factors should include the optical effect of the slanting surfaces on incident light, the effect of vibration on the eye, the restricting effect of acceleration, and the parallax effect of peripheral vision. We do know that visual acuity, or rather, the rapidity of visual perception, is directly related to speed, for the higher the speed, the shorter is the perspective of vision; for example, at 45 miles an hour vision is limited to 200 feet. Thus even if the eyes of an elderly engineer are normal, quickness of perception and reaction time enter the picture, and here of course is where over-all experience counts.

The fourth factor is that of the visual fields. A constricted field of vision, even if unilateral, will have some effect. If bilateral and marked, the result can be disastrous. Arteriosclerosis, syphilis, diabetes, diseases of the blood, and intracranial lesions of all kinds may affect the peripheral and central fields—for example, homonymous hemianopsia. Infections or hemorrhages within the eye may hit the macula and produce a central scotoma. Retinitis pigmentosa causes an extensive peripheral contraction. Toxic causes are to be seen in tobacco-alcohol amblyopia, methyl alcohol poisoning and other chemical injuries to the structure within the eye, optic nerve, or visual cortex. These are but a few examples of lesions that may affect the peripheral vision, so important to us when we are moving rapidly; or the central vision so important to us for our visual acuity.

The fifth factor is that of ocular motility. A strabismus may be either convergent or divergent, it may be constant or intermittent. If constant and of long standing, diplopia is rarely a factor, but if intermittent or paralytic, diplopia may persist. It is obvious, therefore, that our railroader, to be efficient and safe, must not have a significant ocular muscle imbalance. Binocular single vision implies perfect coordination of the two eyes permitting the individual to judge depth and perspective. Retinal motion, ocular muscle coordination, stereopsis, and eye-hand coordination vary together as the flow of perception speeds by, particularly below and on the sides, more swiftly the nearer. Insinuate, therefore, any anomalous

factor into the coordination, and visual efficiency correspondingly drops.

The sixth factor, color vision or sense, is an obvious requirement for the recognition of railroad color signals, so necessary for safety. This has long been recognized and a defect in color vision of the railroad employee who has to rely on colored lights and signals in his work, is sufficient to disqualify him. Congenitally colorblind individuals are easily spotted, but consider the hazard of the employment of a worker who has suddenly acquired color blindness, especially for red and green, which is an early symptom of toxic amblyopia or optic neuritis.

Finally, let us consider briefly the effect of injury or disease of the cornea on visual efficiency. The railroad worker is particularly liable to sustain injuries by foreign bodies of all sorts, often lumped together as "cinders," that strike his cornea and become embedded. Protective goggles have been a wonderful invention and are more and more used, but hundreds of cases of foreign bodies of the cornea are a daily occurrence throughout the railroad industry. Every time a foreign body becomes embedded in the cornea it has two effects. The immediate one produces tearing and pain, which may be so acute that the worker momentarily and instinctively forgets what he is doing. The other one is remote. This is the resultant scar formation. It may be trivial, or it may, if it is situated in the visual axis of the cornea, result in marked visual loss and a permanent decrease in visual efficiency. Diseases such as superficial punctate keratitis, herpes zoster, or ulcers of the cornea may also leave permanent and disturbing scars. Burns, thermal, chemical, and actinic, likewise may lead to corneal scarring that influences visual efficiency.

SUMMARY

Factors that influence the visual efficiency of the railroad worker are visibility, speed, general and ocular health, visual acuity, ocular motility, fusion, fields of vision, and color sense. Ocular injuries and diseases, to which the railroad worker is especially liable, affecting the cornea, are flying particles of all kinds and sizes, chemicals of various sorts, and virus and bacterial diseases of the conjunctiva and cornea.

With increasing age the eye shows many changes that affect visual acuity. The lens is particularly susceptible and becomes sclerosed and more opaque. Diseases of the blood vessels that are concomitant with age affect the retinal vessels and lead to disturbances in the fields of vision, both peripheral and central. For these reasons engineers, particularly those over 50, should have their eyes and visual efficiency investigated and evaluated at frequent intervals.

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SECTION ON INSTRUCTION

FIFTY-SEVENTH ANNUAL MEETING

American Academy of Ophthalmology and Otolaryngology

October 12-17, 1952
PALMER HOUSE
Chicago, Illinois

A. D. RUEDEMANN, M.D.
Secretary for Instruction — Ophthalmology

DEAN M. LIERLE, M.D.
Secretary for Instruction — Otolaryngology and Maxillofacial Surgery

The following condensed schedules of instruction courses are reprinted for the convenience of Fellows who have not yet placed orders for tickets.

Courses already sold out are so designated on the charts. Please do not request tickets for these courses.

For complete listing of course titles, instructors, and descriptive paragraphs about each subject, please refer to the March-April issue of the TRANSACTIONS.

General Instructions

Only members who have paid their 1952 *dues may order instruction tickets in advance of the meeting.* Members should order tickets only for their own use. Candidates for fellowship in the Academy are not privileged to reserve tickets in advance. After the session opens, no distinction between members, candidates and guests will be made in selling tickets.

Each period (1 hour) of instruction costs $2.00. *Orders unaccompanied by properly executed checks will not be filled.* Checks should be made payable to W. L. Benedict, M.D.

In case of inability to attend the meeting after order has been filled, a refund will be granted if requested prior to October 10.

If courses requested are not available and it is impossible to substitute, a refund will be granted. Refunds will not be sent by mail. A blue refund card will be placed in the Advance Registration Envelope which is received at the time of registration. This should be presented at the Instruction Desk for payment.

All orders for tickets will be filled according to postmark on envelope containing check. Telegrams will not be honored. Previous to October 1, send orders to 100 First Avenue Building, Rochester, Minnesota; after October 1, mail orders direct to W. L. Benedict, M.D., c/o Palmer House, Chicago, Illinois. Advance orders will not be filled if received after October 9.

Ordering Instruction Tickets

An *individual* course is complete in one (1) period. Most individual courses are repeated on two or three different days allowing the subscriber to choose the day on which he wishes to attend.

A *continuous* course lasts two (2) or more periods as indicated. It is not possible to purchase a ticket to only *part* of

a continuous course.

At the end of this section is printed a colored sheet to be used when ordering tickets in advance. Specify preferences on this sheet and mail to the executive office. Careful attention to instructions will ensure the correct filling of your requests. *Since number of tickets for each course is limited, be sure to list second and third preferences for each period in case first preference is already sold out. In every case, effort will be made to furnish first choices.*

If you are requesting registration in the continuous courses, please specify preferences for individual courses as substitutes in order to avoid disappointment should the continuous courses which you select be sold out when your order is received.

Acknowledgement of Advance Order

As soon as an advance order is filled, a postcard, listing the courses that have been reserved for him, will be mailed to the subscriber. Please do not ask to change courses after your order has been filled. The postcard is merely an assurance that your order has been filled; it is not necessary for registration at the Academy meeting.

Claiming Instruction Tickets

No tickets will be sent through the mails. Members who have ordered instruction course tickets will register at the *Advance Order Section* of the Registration Desk during the week of the Convention to secure registration badge, tickets, and all convention material.

KEY TO DESIGNATION OF PERIODS

The periods will be designated with the following key: Select tickets for — Otolaryngology, forenoons, periods 1-2-3 — Ophthalmology, afternoons, periods 4-5-6

Day	(M)onday	(T)uesday	(W)ednesday	(Th)ursday
Period a.m.		1 2 3	1 2 3	1 2 3
Period p.m.	4 5 6	4 5 6	4 5 6	4 5 6

M-4	2:00 p.m. to 3:00 p.m.	W-4	2:00 p.m. to 3:00 p.m.
M-5	3:15 p.m. to 4:15 p.m.	W-5	3:15 p.m. to 4:15 p.m.
M-6	4:30 p.m. to 5:30 p.m.	W-6	4:30 p.m. to 5:30 p.m.
T-1	9:00 a.m. to 10:00 a.m.	Th-1	9:00 a.m. to 10:00 a.m.
T-2	10:15 a.m. to 11:15 a.m.	Th-2	10:15 a.m. to 11:15 a.m.
T-3	11:30 a.m. to 12:30 p.m.	Th-3	11:30 a.m. to 12:30 p.m.
T-4	2:00 p.m. to 3:00 p.m.	Th-4	2:00 p.m. to 3:00 p.m.
T-5	3:15 p.m. to 4:15 p.m.	Th-5	3:15 p.m. to 4:15 p.m.
T-6	4:30 p.m. to 5:30 p.m.	Th-6	4:30 p.m. to 5:30 p.m.
W-1	9:00 a.m. to 10:00 a.m.		
W-2	10:15 a.m. to 11:15 a.m.		
W-3	11:30 a.m. to 12:30 p.m.		

(Familiarity with this key will be helpful and will avoid confusion)

CHANGES IN INSTRUCTION COURSE PROGRAM AS LISTED IN THE MARCH-APRIL 1952 TRANSACTIONS

Dr. Wilfred E. Fry of Philadelphia will give the lecture on "Glaucoma Secondary to Uveitis" on W-5 in Course 33, replacing Dr. F. Bruce Fralick.

Dr. E. King Gill will conduct Course 426, "External Otitis." Dr. William D. Gill, deceased, was originally scheduled to deliver this lecture.

Because of the death of Dr. Richard G. Scobee continuous course 42 has been cancelled.

CONTINUOUS COURSES — OPHTHALMOLOGY

COURSE	SPEAKERS	SUBJECT	PERIODS USED
1	Allen, J. H., et al	Bacteriology & Infectious Diseases	8 PERIODS M-4, M-5, T-4, T-5, W-4, W-5, Th-4, Th-5
2	Ascher	Aqueous Veins in Glaucoma	2 PERIODS W-5, W-6
3	Bedell	Medical Ophthalmoscopy	4 PERIODS M-5, T-5, W-5, Th-5
4	Berliner	..... SOLD OUT	
5	Birge, et al	New Theories — Retinal Venous Thrombosis	2 PERIODS Th-4, Th-5
6 or 7	Boeder	Aniseikonia & Space Eikonometry	2 PERIODS W-4, W-5 or Th-4, Th-5
8	Brown, et al	Eye Muscles	8 PERIODS M-4, M-5, T-4, T-5, W-4, W-5, Th-4, Th-5
9	Clark	Surgery to Ocular Muscles	2 PERIODS M-5, M-6
10 or 11	Copeland	Streak Retinoscopy	4 PERIODS M-4, M-5, T-4, T-5 or W-4, W-5, Th-4, Th-5
12	Costenbader, et al	Concomitant Strabismus	12 PERIODS M-4, M-5, M-6, T-4, T-5, T-6, W-4, W-5, W-6, Th-4, Th-5, Th-6
13	DeVoe	Clinical Application of Gonioscopy	2 PERIODS Th-5, Th-6
14	Elwyn	Heredodegenerative Diseases of Retina	2 PERIODS M-5, M-6
15	Fink	Management of Oblique Muscle Problems	3 PERIODS T-5, W-5, Th-5
16	Hardy, et al	Basic Principles of Orthoptics	4 PERIODS M-4, T-4, W-4, Th-4
17	Hastings	Psychosomatic Medicine	2 PERIODS W-5, W-6
18 or 19	Henderson	Perimetry	2 PERIODS M-4, M-5 or Th-4, Th-5
20	Hughes, et al	Plastic Surgery	10 PERIODS M-4, M-5, M-6, T-4, T-5, T-6, W-4, W-5, Th-4, Th-5
21	Jardon	Fitting of Ocular Prostheses	2 PERIODS W-5, W-6
22 or 23	Johnson, L. V.	Aqueous Tonography	2 PERIODS W-5, W-6 or Th-4, Th-5
24 or 25	Johnson, V. C., & Davies	Diagnostic Value of X-ray	2 PERIODS M-5, M-6 or T-5, T-6
26	Knighton, et al	Highlights of Glaucoma	8 PERIODS M-5, M-6, T-5, T-6, W-5, W-6, Th-5, Th-6
27	Kuhn	Industrial Ophthalmology	3 PERIODS M-4, T-4, W-4

CONTINUOUS COURSES — OPHTHALMOLOGY

COURSE	SPEAKERS	SUBJECT	PERIODS USED
28	Lebensohn, et al	Refraction	6 PERIODS W-4, W-5, W-6, Th-4, Th-5, Th-6
29	Lowrey	Military Ophthalmology	2 PERIODS W-5, W-6
30	Lyle	Neuro-Ophthalmology	4 PERIODS M-4, T-4, W-4, Th-4
31	McLean, et al	..... SOLD OUT	
32	Meek	Surgical Anatomy of Orbit	2 PERIODS T-5, T-6
33	Meyer, et al	Glaucoma	8 PERIODS M-4, M-5, T-4, T-5, W-4, W-5, Th-4, Th-5
34	Ogle	Essential Aspects of Binocular Vision	2 PERIODS M-5, M-6
35	O'Rourke	Manifest & Cycloplegic Refraction	2 PERIODS T-5, T-6
36	Paton	Corneal Surgery	3 PERIODS T-4, W-4, Th-4
37	B. Payne, et al	Practical Eye Pathology	8 PERIODS M-4, M-5, T-4, T-5, W-4, W-5, Th-4, Th-5
38	Pino	Bifocal Lenses & Presbyopia	2 PERIODS M-5, M-6
39	Romaine, et al	Ocular Motility	12 PERIODS M-4, M-5, M-6, T-4, T-5, T-6, W-4, W-5, W-6, Th-4, Th-5, Th-6
40 or 41	Schepens	Ophthalmoscopy in Diagnosis of Retinal Detachment	2 PERIODS M-5, M-6 or T-5, T-6
42	Scobee, et al	..... CANCELLED	
43	Stow, et al	Applied Clinical Pathology	8 PERIODS M-4, M-5, T-4, T-5, W-4, W-5, Th-4, Th-5
44	Theobald, et al	Histopathology	12 PERIODS M-4, M-5, M-6, T-4, T-5, T-6, W-4, W-5, W-6, Th-4, Th-5, Th-6
45	Thorpe, et al	Slit Lamp Biomicroscopy	12 PERIODS M-4, M-5, M-6, T-4, T-5, T-6, W-4, W-5, W-6, Th-4, Th-5, Th-6
46	Vail & Minsky	Zonular Membrane & Cataract Surgery	2 PERIODS M-5, M-6
47	Wheeler & Knapp	Concomitant Strabismus	6 PERIODS M-5, M-6, T-5, T-6, W-5, W-6
48	Wiener	Practical Points in Surgery	3 PERIODS M-6, T-6, W-6
49	Friedenwald	Measurement of Aqueous Flow	2 PERIODS W-4, W-5

SCHEDULE OF INDIVIDUAL COURSES — OPHTHALMOLOGY — Monday

Key M-4 — 2:00 p.m. to 3:00 p.m.

Course No.	SPEAKER	SUBJECT
204	Atkinson	Measures in Cataract Extraction
205	Baird	Acute Lesions of Cornea
206	Baker	Neuralgias of Head
212	Byrnes	Adjuncts in Teaching Ophthalmology
219	Cordes	Surgery of Congenital Cataracts
226	Falls	Ocular Geriatrics
235	Gipner	Recent Advances
245	Hogan	Pathology of the Choroid
256	Lloyd	Macular Lesions
262	McCaslin	Ocular Anesthesia
268	Osher	Diagnostic Approach to Squint Surgery
269	Owens & Owens	Retrolental Fibroplasia
272	Pfeiffer	Diagnosis with Aid of X-ray
274	Post, L. T.	Difficult Problems in Refraction
275	Post, M. H.	Operating Room Technic
280	Rychener	Lesions of the Lids
285	Spaeth	Lacrimal Gland & Sac
297	Whalman	Minor Surgical Procedures of Eye
A	Home Study	Embryology, Anatomy & Neuro-Anatomy

Key M-5 — 3:15 p.m. to 4:15 p.m.

Course No.	SPEAKER	SUBJECT
207	Barkan	Congenital Glaucoma
209	Berens	Uveitis: Etiology & Treatment
220	Cusick	Ptosis
224	Dunnington	Ocular Wound Healing
229	Fread	Ideal Refraction Equipment
252	Leinfelder	Papilledema & Optic Neuritis
255	Linksz	Visual Physiology
266	Newell	Rabbit Surgery
278	Rudolph	Advantages of General Anesthesia
290	Swan	Value of New Drugs
294	Veasey	Refraction Difficulties
B	Home Study	Physiology

Key M-6 — 4:30 p.m. to 5:30 p.m.

Course No.	SPEAKER	SUBJECT
201	Alvaro	Uveitis: Etiology & Treatment
203	Amenabar	Detachment of Retina: Treatment
216	Chace	Ocular Malingering
222	Davies	Clinical Pathology in Optic Atrophy
223	Dunlap	Divergence Anomalies
231	Fry	Differential Diagnosis of Lesions at Posterior Pole of Eye
233	Gemeroy	External Diseases of Eye
239	Gray	Intraocular Foreign Bodies
243	Hanser	Ocular Allergy
253	Lemoine	Vitreous Opacities
254	Lepard	Eye Diseases Common to Children
261	Marshall	Lid Infections
270	Payne, F.	Unilateral Visual Failure
277	Reese	Cataract Extraction
279	Rundles	Streak Retinoscopy
281	Scheie	Surgery in Congenital Glaucoma
C	Home Study	Optics & Principles of Biomicroscopy

SCHEDULE OF INDIVIDUAL COURSES — OPHTHALMOLOGY — Tuesday

Key T-4 — 2:00 p.m. to 3:00 p.m.

Course No.	SPEAKER	SUBJECT
202	Alvis	End Results of Cataract Wound Closure
204	Atkinson	Measures in Cataract Extraction
205	Baird	Acute Lesions of Cornea
206	Baker	Neuralgias of Head
217	Chandler	Secondary Glaucoma in Aphakia
228	Fralick	External Ocular Manifestations of General Disease
235	Gipner	Recent Advances
240	Guibor	Physiology of Ocular Muscles
256	Lloyd	Macular Lesions
257	Lueck	Practical Optics
262	McCaslin	Ocular Anesthesia
269	Owens & Owens	Retrolental Fibroplasia
272	Pfeiffer	Diagnosis with Aid of X-ray
275	Post, M. H.	Operating Room Technic
283	Siniscal	Chronic Lid Conditions
286	Spaeth	Plastic Surgery
297	Whalman	Minor Surgical Procedures of Eye
D	Home Study	Refraction

Key T-5 — 3:15 p.m. to 4:15 p.m.

Course No.	SPEAKER	SUBJECT
209	Berens	Uveitis: Etiology & Treatment
210	Berke	Surgery of Oblique Muscles
220	Cusick	Ptosis
224	Dunnington	Ocular Wound Healing
227	Falls	Practical Application of Heredity
229	Fread	Ideal Refraction Equipment
252	Leinfelder	Papilledema & Optic Neuritis
255	Linksz	Visual Physiology
276	Randolph	Handling Patients with Advanced Glaucoma
282	Simpson	Convergence Insufficiency & Divergence Excess: Office Management
291	Tait	Methods in Correction of Accommodation & Convergence Abnormalities
294	Veasey	Refraction Difficulties
296	Walsh	Interesting Neuro-Ophthalmologic Cases
E	Home Study	External Diseases, Bacteriology & Virology

Key T-6 — 4:30 p.m. to 5:30 p.m.

Course No.	SPEAKER	SUBJECT
201	Alvaro	Uveitis: Etiology & Treatment
203	Amenabar	Detachment of Retina: Treatment
216	Chace	Ocular Malingering
222	Davies	Clinical Pathology in Optic Atrophy
223	Dunlap	Divergence Anomalies
231	Fry	Differential Diagnosis of Lesions at Posterior Pole of Eye
233	Gemeroy	External Diseases of Eye
239	Gray	Intraocular Foreign Bodies
243	Hanser	Ocular Allergy
246	Iliff	Beta Irradiation Therapy
253	Lemoine	Vitreous Opacities
261	Marshall	Lid Infections
270	Payne, F.	Unilateral Visual Failure
273	Pischel	Retinal Detachment Surgery: Complications
279	Rundles	Streak Retinoscopy
281	Scheie	Surgery in Congenital Glaucoma
287	Steffensen	ACTH & Cortisone
F	Home Study	Perimetry & Neuro-Ophthalmology

SCHEDULE OF INDIVIDUAL COURSES — OPHTHALMOLOGY — Wednesday

Key W-4 — 2:00 p.m. to 3:00 p.m.

Course No.	SPEAKER	SUBJECT
202	Alvis	End Results of Cataract Wound Closure
211	Bettman	Value of Goniotomies
213	Calhoun	Dystrophies & Degenerations of Cornea
217	Chandler	Secondary Glaucoma in Aphakia
221	Cutler	Procedures Used in Enucleation
238	Gordon	Anatomy of Orbital Fascia
241	Guibor	Neuromotor Defects of Ocular Muscles
244	Harrington	Psychosomatic Phases
247	Jobe	Practical Physiologic Optics
263	McGuire	Heterophoria & Heterotropia
271	Perera	Retinal Detachment
283	Siniscal	Chronic Lid Conditions
284	Smith	Practical Points in Office Treatment
292	Theodore	Ocular Therapeutics
298	Wiener	Minor Lid Surgery
G	Home Study	Extraocular Muscles

Key W-5 — 3:15 p.m. to 4:15 p.m.

Course No.	SPEAKER	SUBJECT
208	Bellows	Changes in Crystalline Lens
210	Berke	Surgery of Oblique Muscles
215	Cassady	Dacryocystitis of Infancy
232	Gailey	Complications of Cataract Surgery
234	Gifford	Dacryocystorhinostomy
237	Goar	Dystrophies of Cornea
249	Klien	Clinical Pathology of Retinal Lesions
264	Morrison	Office Management of Glaucoma
267	Olson	Iritis, Iridocyclitis, & Keratitis
291	Tait	Methods in Correction of Accommodation & Convergence Abnormalities
293	Trueman	Cerebral Lesions
H	Home Study	Ophthalmoscopy & Medical Ophthalmology

Key W-6 — 4:30 p.m. to 5:30 p.m.

Course No.	SPEAKER	SUBJECT
214	Callahan	Repair of Lid Lacerations
218	Cholden	Informing Patient of Blindness: Psychiatric Aspects
225	Egan	Cross Cylinder Application & Theory
236	Givner	Systemic Disease
246	Iliff	Beta Irradiation Therapy
248	Katz	Corneal Dystrophies
250	Krewson	Clinical Significance of Oblique Muscles
251	Kronfeld	End Results in Glaucoma Surgery
254	Lepard	Eye Diseases Common to Children
258	Macnie	Head Trauma with Ocular Significance
259	Mangold	Proper Fitting of Glasses
260	Mann, M.	Hypertensive Changes in Fundus
265	Mullen	Ophthalmic Emergencies
287	Steffensen	ACTH & Cortisone
288	Stimson	Lenses for the Aphakic
299	Mann, M.	Refraction and Lens for Cataract Patient
I	Home Study	Pathology

SCHEDULE OF INDIVIDUAL COURSES — OPHTHALMOLOGY — Thursday

Key Th-4 — 2:00 p.m. to 3:00 p.m.

Course No.	SPEAKER	SUBJECT
211	Bettman	Value of Goniotomies
213	Calhoun	Dystrophies & Degenerations of Cornea
221	Cutler	Procedures Used in Enucleation
238	Gordon	Anatomy of Orbital Fascia
242	Guibor	Neurology of Ocular Muscles
244	Harrington	Psychosomatic Phases
247	Jobe	Practical Physiologic Optics
263	McGuire	Heterophoria & Heterotropia
267	Olson	Iritis, Iridocyclitis, & Keratitis
271	Perera	Retinal Detachment
284	Smith	Practical Points in Office Treatment
292	Theodore	Ocular Therapeutics
J	Home Study	Pharmacology & Therapeutics

Key Th-5 — 3:15 p.m. to 4:15 p.m.

Course No.	SPEAKER	SUBJECT
208	Bellows	Changes in Crystalline Lens
215	Cassady	Dacryocystitis of Infancy
232	Gailey	Complications of Cataract Surgery
234	Gifford	Dacryocystorhinostomy
249	Klien	Clinical Pathology of Retinal Lesions
258	Macnie	Head Trauma with Ocular Significance
264	Morrison	Office Management of Glaucoma
293	Trueman	Cerebral Lesions

Key Th-6 — 4:30 p.m. to 5:30 p.m.

Course No.	SPEAKER	SUBJECT
214	Callahan	Repair of Lid Lacerations
225	Egan	Cross Cylinder Application & Theory
236	Givner	Systemic Disease
248	Katz	Corneal Dystrophies
250	Krewson	Clinical Significance of Oblique Muscles
259	Mangold	Proper Fitting of Glasses
260	Mann, M.	Hypertensive Changes in Fundus
265	Mullen	Ophthalmic Emergencies
287	Steffensen	ACTH & Cortisone
288	Stimson	Lenses for the Aphakic
299	Mann, W.	Refraction and Lens for Cataract Patients

SECTION ON INSTRUCTION

ADVANCE ORDER SHEET

All courses cost $2.00 for each period (one hour). Follow instructions explicitly and check for errors in listing before mailing.

Previous to OCTOBER 1

Send order to . . .

WILLIAM L. BENEDICT, M.D., *Executive Secretary-Treasurer*
American Academy of Ophthalmology and Otolaryngology
100 First Avenue Building, Rochester, Minnesota

After OCTOBER 1, mail to . . .

WILLIAM L. BENEDICT, M.D., *Executive Secretary-Treasurer*
American Academy of Ophthalmology and Otolaryngology
c/o Palmer House, Chicago, Illinois

Academy Candidates and Guests May Purchase Tickets at Registration Desk Only

(Over)

ADVANCE ORDER SHEET

ORDERS FILLED ONLY IF CURRENT DUES ARE PAID

NO REFUNDS WILL BE MADE AFTER OCTOBER 10

(Type or Print) Last Name First Name Middle Initial

Office Address

City Zone State

Days to be Present

☐ Monday

☐ Tuesday

☐ Wednesday

☐ Thursday

Total Amount Enclosed $_____

SELECTION OF COURSES ON REVERSE SIDE OF THIS BLANK

RECAPITULATION OF GENERAL INSTRUCTIONS

1. Monday, Tuesday, Wednesday, and Thursday are represented by the letters M, T, W, Th. The first period in the morning is represented by figure 1, second period by figure 2 and third period by figure 3. The first period in the afternoon is represented by figure 4, second period by figure 5, and third period by figure 6. All courses in otolaryngology and maxillofacial surgery are presented in the mornings. All courses in ophthalmology are presented in the afternoons.
2. Reservation of instruction tickets in advance of the meeting will be made ONLY for MEMBERS whose 1952 dues have been paid. Academy candidates and guests may make selection of courses ONLY at the General Registration Desk in Chicago.
3. Make check payable to W. L. Benedict, M.D.
4. Mail order promptly to ensure reservation of first choices. If necessary, an attempt will be made to rearrange schedule to include "first choices."

(Over)

SELECTION OF COURSES

PERIOD		FIRST CHOICE		SECOND CHOICE		THIRD CHOICE	
		Course No.	Lecturer	Course No.	Lecturer	Course No.	Lecturer
MON.	4						
P.M.	5						
	6						
TUES.	1						
A.M.	2						
	3						
P.M.	4						
	5						
	6						
WED.	1						
A.M.	2						
	3						
P.M.	4						
	5						
	6						
THURS.	1						
A.M.	2						
	3						
P.M.	4						
	5						
	6						

CONTINUOUS COURSES — OTOLARYNGOLOGY

COURSE	SPEAKERS	TITLE	PERIODS USED
101	Altmann & Waltner	Histopathology of Ear—Part I	2 PERIODS T-1, T-2
102	Altmann & Waltner	Histopathology of Ear—Part II	2 PERIODS W-1, W-2
103 or 104	Bryan & Bryan	Cytologic Pathology of ENT Diseases	2 PERIODS T-1, T-2 or W-1, W-2
105 or 106	Conley	Cancer of Head & Neck	2 PERIODS T-1, T-2 or Th-2, Th-3
107	Converse	Nasal Plastic Surgery (Basic)	2 PERIODS W-1, W-2
108	Converse	Nasal Plastic Surgery (Advanced)	2 PERIODS Th-2, Th-3
109 or 110	Figi	Lesions of Mouth	2 PERIODS T-2, T-3 or W-2, W-3
111	Fowler & Montreuil	Histology of Ear — Vertical Sections	2 PERIODS T-1, T-2
112	Fowler & Montreuil	Histology of Ear — Horizontal Sections	2 PERIODS Th-1, Th-2
113 or 114	Hansel	Allergy	2 PERIODS T-1, T-2 or Th-1, Th-2
115	Jackson	Technic in Peroral Endoscopy	3 PERIODS T-3, W-3, Th-2
116	Kazanjian, et al	Traumatic Injuries of Face	2 PERIODS T-1, T-2
117 or 118 or 119	Lempert	Evaluating Degree of Hearing Function	2 PERIODS T-2, T-3 or W-2, W-3 or Th-2, Th-3
120 or 121	Lindsay	Vertigo	2 PERIODS W-2, W-3 or Th-2, Th-3
122 or 123	Maliniac	Maxillofacial Plastic Surgery	2 PERIODS T-1, T-2 or W-2, W-3
124	Marcus, et al	Hearing Problems in Children	2 PERIODS W-1, W-2
125	Moses	Speech & Voice Therapy	2 PERIODS W-2, W-3
126 or 127	Mueller	Practical Audiometry	2 PERIODS W-2, W-3 or Th-1, Th-2
128 or 129	Orton	Applied Surgery of Neck	2 PERIODS T-2, T-3 or Th-2, Th-3
130 or 131	Sellers	Equilibration	2 PERIODS T-1, T-2 or Th-2, Th-3
132 or 133	Semenov	Therapy in Pathology of ENT	2 PERIODS T-2, T-3 or Th-2, Th-3
134	Tucker, et al	Esophageal Diseases & Foreign Bodies	3 PERIODS T-1, W-3, Th-1
135	Tucker, et al	Laryngeal Surgery	3 PERIODS T-3, W-2, Th-2
136 or 137	Turchik	Nasal Obstruction	2 PERIODS W-1, W-2 or Th-2, Th-3

SCHEDULE OF INDIVIDUAL COURSES — OTOLARYNGOLOGY — Tuesday

Key T-1 — 9:00 a.m. to 10:00 a.m.

Course No.	SPEAKER	SUBJECT
401	Alexander	Adenoid Operation: Relation to Auditory Apparatus
403	Baron	Modified Radical Mastoidectomy
406	Boyden	Chronic Frontal Sinus Surgery
414	Craft	Nasal Allergy
417	Day	What the Otologist Can Do for Deafness
421	Fabricant	Modern Medication of Nose & Sinuses
426	Gill	External Otitis
427	Glick	Surgical Approaches on Neck
429	Goldman	Rhinologic Diagnosis & Treatment
439	Holinger	Esophageal Diseases in Infants & Children
441	Hosler	Cardiac Arrest During Operation
442	House	Surgery of Chronically Discharging Ear
445	Hunnicutt	When & How to Make Tissue Examination
451	Kemler	Mirror Laryngology
460	Maxwell	Tumors of Face & Neck in Infancy & Childhood
462	McLaurin	Otitis Externa: Therapy
475	Reger	Auditory Malingering
477	Richards	Suggestions Concerning Office Practice
482	Seltzer	Plastic Surgery of Nose & Face-Lift Operation
485	Shea	Intranasal Surgery
491	Tremble	Nose & Throat Medication
497	Wilkerson	Hearing & Speech Center for Your City

Key T-2 — 10:15 a.m. to 11:15 a.m.

Course No.	SPEAKER	SUBJECT
402	Ashley	Dust Therapy in Respiratory Allergy
404	Becker	Plastic Surgery of Protruding Ears
407	Canfield	Audiology for Practicing Otologist
415	Croushore	Hemorrhage: Prevention & Control
422	Farrior & Bagby	Surgical Anatomy of Ear
425	Friedberg	Diseases of Larynx
431	Hallberg	Nosebleed & Its Management
444	Hunnicutt	Chronically Congested Nose
453	Kirchner	Difficulty in Swallowing
464	Miller	Laryngeal Emergencies in Infants & Children
468	Ogura	Laryngectomy & Neck Dissection
470	Pearlman	Tonsil & Adenoid Operation
471	Perlman	Serous Otitis
474	Putney	Surgery of Neck Tumors
480	Ryan	Headache
487	Simmons	Intranasal Dacryocystorhinostomy
489	Singleton	Tonsil & Adenoid Problem in Children
493	Van Alyea	Embryology of ENT
496	Walsh	Otitis Media: Treatment
501	Panel	Otological Problems

Key T-3 — 11:30 a.m. to 12:30 p.m.

Course No.	SPEAKER	SUBJECT
405	Bellucci	Submucous Resection of Nasal Septum
409	Cohen	Deviated Noses & Their Correction
411	Conley	Pigmented Mole & Malignant Melanoma: Treatment
412	Costen	Mandibular Joint Syndrome
416	Cullen	Anesthesia
423	Farrior & Carron	Fenestration Operation
424	Fisher	Esophageal Obstruction
428	Glorig	Evaluation of Methods for Measuring Auditory Acuity
430	Goodhill	Management of Tinnitus
432	Harrill	Bronchography
433	Hilger	Therapeutic Approaches to Inner Ear
438	Holinger	Pediatric Laryngology
443	Howard	Office Procedures in ENT
448	Juers	Hearing Tests
449	Keaster	Speech & Hearing in Children Under Five
458	Lewin	Facial & Nasal Reconstructive Surgery
459	Long	Chemotherapeutic & Antibiotic Agents
466	Morrison	Technic of Tonsillectomy
469	Pastore	A Lump in the Throat
473	Pressman	Rhinologist & Problem of Rhinoplasty
481	Sataloff	Treatment of Nerve Deafness
484	Shambaugh	Failures in Fenestration Surgery
490	Thornell	Intralaryngeal Arytenoidectomy
494	Van Alyea	Chronic Sinusitis

SCHEDULE OF INDIVIDUAL COURSES — OTOLARYNGOLOGY — Wednesday

Key W-1 — 9:00 a.m. to 10:00 a.m.

Course No.	SPEAKER	SUBJECT
401	Alexander	Adenoid Operation: Relation to Auditory Apparatus
403	Baron	Modified Radical Mastoidectomy
408	Carhart	Audiogram in Otologic Diagnosis
413	Cottle	Septal Reconstruction Surgery
414	Craft	Nasal Allergy
417	Day	What the Otologist Can Do for Deafness
422	Farrior & Bagby	Surgical Anatomy of Ear
426	Gill	External Otitis
427	Glick	Surgical Approaches on Neck
429	Goldman	Rhinologic Diagnosis & Treatment
440	Holmes	Scars of Face
441	Hosler	Cardiac Arrest During Operation
450	Kelemen	Mechanical Injuries of Hearing Organ
451	Kemler	Mirror Laryngology
456	LeJeune	Technic of Intralaryngeal Surgery
460	Maxwell	Tumors of Face & Neck in Infancy & Childhood
461	McConnell	Speech Audiometry for Otologist
462	McLaurin	Otitis Externa: Therapy
463	McNally	Labyrinthine Tests
467	Neivert	Care of Instruments
476	Reger	Practical Office Audiometry
477	Richards	Suggestions Concerning Office Practice
479	Roberts, S. E.	Head & Neck Syndrome
483	Senturia	External Otitis
495	Von Leden	Tracheotomy
500	Panel	Tumors & Infections of Neck
503	Panel	Rhinology

Key W-2 — 10:15 a.m. to 11:15 a.m.

Course No.	SPEAKER	SUBJECT
404	Becker	Plastic Surgery of Protruding Ears
407	Canfield	Audiology for Practicing Otologist
415	Croushore	Hemorrhage: Prevention & Control
420	Erich	Fractures of Facial Bones
431	Hallberg	Nosebleed & Its Management
433	Hilger	Therapeutic Approaches to Inner Ear
435	Hill	Antro-Alveolar Fistulas
444	Hunnicutt	Chronically Congested Nose
447	Jordan	Otologic Conditions Complicated by Allergy
452	Kinney	Acoustic Trauma
455	Kuhn	Allergic Diseases of Ear
457	Lenz	Cancer of Nose & Throat: Radiotherapy
466	Morrison	Technic of Tonsillectomy
468	Ogura	Laryngectomy & Neck Dissection
470	Pearlman	Tonsil & Adenoid Operation
472	Pohlman	Plastic Ear Drum
488	Simonton	Recruitment of Loudness & Diplacusis Binauralis
490	Thornell	Intralaryngeal Arytenoidectomy
493	Van Alyea	Embryology of ENT
499	Panel	Diseases of Larynx, Neck, Tracheobronchial Tree & Esophagus

Key W-3 — 11:30 a.m. to 12:30 p.m.

Course No.	SPEAKER	SUBJECT
405	Bellucci	Submucous Resection of Nasal Septum
409	Cohen	Deviated Noses & Their Correction
418	Derlacki	Allergy of Ear
419	DeWeese	The Dizzy Patient
424	Fisher	Esophageal Obstruction
425	Friedberg	Diseases of Larynx
437	Hitschler	Cardiac Arrest & Resuscitation
438	Holinger	Pediatric Laryngology
443	Howard	Office Procedures in ENT
448	Juers	Hearing Tests
449	Keaster	Speech & Hearing in Children Under Five
454	Kos	Clinical Orientation in Management of Dizziness
459	Long	Chemotherapeutic & Antibiotic Agents
465	Moore	Diseases of Maxillary Sinus
475	Reger	Auditory Malingering
478	Roberts, G. J.	Management of Nasal Fractures
486	Shuster	Ocular Conditions of Otologic & Nasal Origin
491	Tremble	Nose & Throat Medication
492	Tschiassny	Locating Lesion in Facial Nerve Paralysis
497	Wilkerson	Hearing & Speech Center for Your City
498	Panel	Head Pain
501	Panel	Otological Problems
502	Panel	Medications

SCHEDULE OF INDIVIDUAL COURSES — OTOLARYNGOLOGY — Thursday

Key Th-1 — 9:00 a.m. to 10:00 a.m.

Course No.	SPEAKER	SUBJECT
406	Boyden	Chronic Frontal Sinus Surgery
408	Carhart	Audiogram in Otologic Diagnosis
410	Cohen	Rhinoplasty Complications
421	Fabricant	Modern Medication of Nose & Sinuses
423	Farrior & Carron	Fenestration Operation
432	Harrill	Bronchography
440	Holmes	Scars of Face
442	House	Surgery of Chronically Discharging Ear
445	Hunnicutt	When & How to Make Tissue Examination
455	Kuhn	Allergic Diseases of Ear
456	LeJeune	Technic of Intralaryngeal Surgery
461	McConnell	Speech Audiometry for Otologist
463	McNally	Labyrinthine Tests
471	Perlman	Serous Otitis
476	Reger	Practical Office Audiometry
479	Roberts, S. E.	Head & Neck Syndrome
480	Ryan	Headache
481	Sataloff	Treatment of Nerve Deafness
483	Senturia	External Otitis
485	Shea	Intranasal Surgery
486	Shuster	Ocular Conditions of Otologic & Nasal Origin
488	Simonton	Recruitment of Loudness & Diplacusis Binauralis
492	Tschiassny	Locating Lesion in Facial Nerve Paralysis
495	Von Leden	Tracheotomy
503	Panel	Rhinology

Key Th-2 — 10:15 a.m. to 11:15 a.m.

Course No.	SPEAKER	SUBJECT
402	Ashley	Dust Therapy in Respiratory Allergy
413	Cottle	Septal Reconstruction Surgery
416	Cullen	Anesthesia
420	Erich	Fractures of Facial Bones
434	Hilger	Facial Fractures
436	Hill	Aural Rehabilitation
446	Jones	Tear Sac Surgery
447	Jordan	Otologic Conditions Complicated by Allergy
453	Kirchner	Difficulty in Swallowing
457	Lenz	Cancer of Nose & Throat: Radiotherapy
464	Miller	Laryngeal Emergencies in Infants & Children
467	Neivert	Care of Instruments
472	Pohlman	Plastic Ear Drum
489	Singleton	Tonsil & Adenoid Problem in Children
496	Walsh	Otitis Media: Treatment
499	Panel	Diseases of Larynx, Neck, Tracheobronchial Tree & Esophagus

Key Th-3 — 11:30 a.m. to 12:30 p.m.

Course No.	SPEAKER	SUBJECT
412	Costen	Mandibular Joint Syndrome
418	Derlacki	Allergy of Ear
419	DeWeese	The Dizzy Patient
428	Glorig	Evaluation of Methods for Measuring Auditory Acuity
430	Goodhill	Management of Tinnitus
439	Holinger	Esophageal Diseases in Infants & Children
450	Kelemen	Mechanical Injuries of Hearing Organ
452	Kinney	Acoustic Trauma
454	Kos	Clinical Orientation in Management of Dizziness
458	Lewin	Facial & Nasal Reconstructive Surgery
465	Moore	Diseases of Maxillary Sinus
469	Pastore	A Lump in the Throat
473	Pressman	Rhinologist & Problem of Rhinoplasty
474	Putney	Surgery of Neck Tumors
478	Roberts, G. J.	Management of Nasal Fractures
482	Seltzer	Plastic Surgery of Nose & Face-Lift Operation
484	Shambaugh	Failures in Fenestration Surgery
494	Van Alyea	Chronic Sinusitis
498	Panel	Head Pain
500	Panel	Tumors & Infections of Neck
502	Panel	Medications

News - - - Notes

INTERNATIONAL ASSOCIATION OF MEDICAL PRESS

The International Association of Medical Press will hold a Pan-American Congress of the Medical Press in Buenos Aires, Argentina, on July 12 to 16, 1953. Dr. Morris Fishbein is president of the association.

PAN-AMERICAN ASSOCIATION OF OPHTHALMOLOGY

Dr. Alston Callahan, professor of ophthalmology at the Medical College of Alabama, recently visited South America, his tour being sponsored by the Pan-American Association of Ophthalmology. Dr. Callahan visited Lima and then flew to Sao Paulo, where he gave two lectures at the Centro de Estudos de Ophthalmologia on May 21 and 23. His subjects were "Reparative Surgery of the Eyelids" and "Management of Penetrating and Blunt Injuries of the Eye," respectively. He went on to Rio, from Rio to Caracas, Venezuela, and thence back to the United States.

NEW ENGLAND OTO-LARYNGOLOGICAL SOCIETY

The regular Spring Meeting of the New England Oto-Laryngological Society was held at the Massachusetts Eye and Ear Infirmary in Boston on June 18. Dr. LeRoy A. Schall presiding. Among the speakers were Drs. Francis Lowell, Francis L. Weille, Maurice G. Evans, Walter B. Hoover, Peter Knapp, and Philip E. Meltzer.

The next meeting of the society will be held on Wednesday, November 19, 1952, and the annual meeting for 1953 will be held in conjunction with the Eastern Section of the American College of Surgeons on March 3 and 4, 1953.

CALIFORNIA

The Los Angeles Society of Ophthalmology and Otolaryngology held a dinner meeting on June 20. Taking part in the program were Dr. Bert W. Meyer, who discussed "Prevention and Treatment of Cardiac Arrest," Dr. Herman Semenov, whose subject was "Intranasal Use and Abuse of Cortisone," Dr. Walter H. Martin, who read a paper on "Methods of Treating Persistent Tympanic Perforation," and Dr. Manuel Wexler, the subject of whose paper was "Rhinoplastic Surgery Complications."

Dr. W. H. Hollinshead, Professor of Anatomy at the Mayo Foundation, Rochester, Minn., was the guest speaker at the Joint Meeting of the Los Angeles Society of Ophthalmology and Otolaryngology and the Los Angeles Ophthalmological Society on June 5, 1952. Dr. Hollinshead presented his motion picture, "Anatomy of the Orbit and Adjacent Structures." The film, which is in color, shows the dissection of the lids and orbit from the front, as well as related structures about the chiasm.

NEW YORK

The Third Annual Graduate Convention of the Society of Ex-House Surgeons of the Manhattan Eye, Ear, and Throat Hospital was held at 210 East 64th St., New York 21, N. Y. on April 21, 22 and 23, 1952. About forty doctors were on the lecture and operative program.

Of those attending the Convention, the ophthalmologists outnumbered the otolaryngologists three to one.

NORTH CAROLINA SOUTH CAROLINA

There will be a joint meeting of the North Carolina Eye, Ear, Nose, and Throat Society and the South Carolina Society of Ophthalmology and Otolaryngology in Winston-Salem, N. C. on September 15 to 17, 1952. Headquarters will be the Robert E. Lee Hotel in Winston-Salem.

Guest ophthalmologists who will be present are Dr. P. Robb McDonald and Dr. Irving H. Leopold of Philadelphia, Dr. Harold W. Brown of New York City, and Dr. Frank W. Newell of Chicago. The following otolaryngologists will be on the program: Dr. Theodore Walsh of St. Louis and Dr. Frank Turchik of Bridgeport, Conn. A third otolaryngologist will be announced at a later date.

Dr. G. B. Ferguson, The McPherson Hospital, 1110 West Main Street, Dur-

ham, N. C., is secretary of the North Carolina Eye, Ear, Nose, and Throat Society, and Dr. Roderick Macdonald, 330 East Main Street, Rock Hill, S. C., is secretary of the South Carolina Society of Ophthalmology and Otolaryngology.

PENNSYLVANIA

Dr. Louis H. Clerf, Professor of the Department of Laryngology and Broncho-Esophagology of Jefferson Medical College, Philadelphia, spoke on "Laryngeal Lesions of Epithelial Origin" at a meeting of the Reading Eye, Ear, Nose and Throat Society on April 16.

VIRGINIA

The Twenty-fifth Annual Spring Congress of the Gill Memorial Eye, Ear and Throat Hospital was held in Roanoke, Va., April 7 to 12, 1952. The total registration including ladies was 464. Next year the meeting will be held April 6 through 11.

CANADA

The Faculty of Medicine of the University of Toronto will offer a Refresher Course in Eye Surgery from April 13 to 17, 1953. The instruction will consist of lectures, operative clinics on patients and cadaver surgery in small groups.

The guest surgeons will be Dr. Dohrmann K. Pischel, San Francisco, and Mr. H. B. Stallard, F.R.C.S., London, England.

The staff of the Department of Ophthalmology in the University will contribute extensively to the course. The course will be given for a minimum of 10 and a maximum of 30 students. The fee will be $100.00 payable to the Chief Accountant, Simcoe Hall, University of Toronto. Applications should be made to the Dean of the Faculty of Medicine, not later than January 31, 1953.

The University of Toronto, Faculty of Medicine, offers a postgraduate course in ophthalmology extending over three years. The graduate instruction in ophthalmology in the teaching hospitals in Toronto has been coordinated under the direction of the University. The first year on a Fellowship the student spends in one of the basic sciences of ophthalmology, and the final two years are spent on the intern service of one or more of the University teaching hospitals.

The fee for instruction is $50 per year payable to the Chief Accountant, University of Toronto. An application for appointment may be made to the professor of ophthalmology, Faculty of Medicine, University of Toronto. Appointments are made in December to commence on the following July 1.

POSITIONS AVAILABLE

Residencies in Ophthalmology and Otolaryngology in a recognized teaching hospital with an active out-patient clinic and a sufficient number of in-patients for residents to get good training in the surgical and medical care of hospital patients. In May an affiliated Eye, Ear, Nose, and Throat Hospital was opened and additional residents are needed to help staff this new hospital. All teaching will be done by certified members of the faculty of the medical school. Will consider combined residency applicant. Starting salary, $1200 yearly plus maintenance. Apply to Director of Personnel, Medical College of Virginia, Richmond.

Listings of positions available should be sent to W. L. Benedict, M.D., 100 First Avenue Building, Rochester, Minn., by the fifth of the month preceding publication. They should include (1) type of physician wanted (ophthalmologist or otolaryngologist; diplomate, resident, etc.); (2) type of position to be filled; and (3) whom to write for further information. Unless otherwise requested, listings will be published once only.

The Academy will handle no further correspondence beyond the listing and assume no responsibility. Neither does it endorse or guarantee any of the published listings.

Residency in Ophthalmology beginning July 1, 1952 at the George F. Geisinger Memorial Hospital and Foss Clinic, Danville, Pa. Applicant must have had previous ophthalmic training and/or postgraduate course in ophthalmology. Statistics for 1951: Out patients, 11,192; refractions, 5,480; consultations, 394; ophthalmic operations, 362; glasses released, 3,551; cataract extractions, 78; ophthalmic admissions, 285. Applications should be addressed to Clyde H. Jacobs, M.D., Director of the Department of Ophthalmology, Foss Clinic, Danville, Pa.

Position as associate to certified otolaryngologist also doing endoscopy with excellent volume of practice. Association with group of seven men, two certified internists, certified surgeon, certified ophthalmologist and two general practitioners. Opportunity for investment in physical assets if desired or earning partnership in practice. Prefer certified man preferably under 35 years of age. Write Dr. A. L. Frye, Sharon Medical Clinic, Sharon, Pa.

DIRECTORY OF OPHTHALMOLOGIC AND OTOLARYNGOLOGIC SOCIETIES

INTERNATIONAL

INTERNATIONAL ASSOCIATION FOR PREVENTION OF BLINDNESS

President: Dr. P. Bailliart, 47 Rue de Bellechasse, Paris, France
Secretary-General: Prof. A. Franceschetti, rue A—Jentzer 22, Geneva, Switzerland

INTERNATIONAL COUNCIL OF OPHTHALMOLOGY

President: Prof. F. W. Nordenson
Secretary: Dr. Holger Ehlers, Rigshospital, Copenhagen, Denmark

INTERNATIONAL ORGANIZATION AGAINST TRACHOMA

President: Dr. A. F. MacCallan
Secretary: Dr. F. Wibaut, P. C. Hoofstraat 145, Amsterdam, Holland

PAN-AMERICAN ASSOCIATION OF OPHTHALMOLOGY

President: Dr. M. E. Alvaro
Secretary: Dr. Thomas D. Allen, 122 South Michigan Avenue, Chicago 2, Ill.
Time and Place: February 1956. Santiago, Chile

PAN-AMERICAN ASSOCIATION OF OPHTHALMOLOGY, PUERTO RICO CHAPTER

President: Dr. Luis J. Fernandez, Box 2206, San Juan 10, Puerto Rico
Secretary: Dr. P. Fernandez
Place: San Juan

PAN-AMERICAN ASSOCIATION OF OTO-RHINO-LARYNGOLOGY AND BRONCHO-ESOPHAGOLOGY

President: Dr. Jose C. Gros
Secretary: Dr. Pedro Hernandez Gonzalo, Calle 8, 358, Havana, Cuba

FOREIGN

THE OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY OF ALBERTA

President: Dr. W. E. Spankie
Secretary: Dr. M. R. Marshall, Wells Pavilion, University Hospital, Edmonton, Alberta, Canada
Time and Place: Spring of 1953 in Edmonton

ALL-INDIA OPHTHALMOLOGICAL SOCIETY

President: Dr. G. Zachariah
Secretaries: Dr. S. N. Cooper and Dr. V. K. Chitnis, Laud Mansion, Queen's Road, Bombay 4, India

ASSOCIACAO MEDICA DO INSTITUTO PENIDO BURNIER

President: Dr. Leoncio de Souza ueiroz
First Secretary: Dr. Milton B. de Toledo
Second Secretary: Dr. Aloisio Af. Ferreira

AUSTRIAN OTOLARYNGOLOGICAL SOCIETY, VIENNA

President: Prof. Dr. E. Schlander
Secretary: Doz. Dr. O. Novotny, Vienna IX, Alserstrasse 4, Austria
Time: Annually

BELGIAN OPHTHALMOLOGIC SOCIETY

President: Dr. L. Weekers
Secretary-General: Dr. M. Appelmans, Avenue Ruelens 179, Louvain
Time and Place: January, June and November. Bruxelles

BOMBAY OPHTHALMOLOGISTS' ASSOCIATION

Chairman: Rotated
Conveners: Dr. S. N. Cooper and Dr. B. D. Telang, Laud Mansion 21, Queen's Road, Bombay 4
Time and Place: 7:30 p.m., third Wednesday of each month. The seven ophthalmic hospitals of Bombay by rotation

BRITISH ASSOCIATION OF OTOLARYNGOLOGISTS

President: Mr. V. E. Negus
Honorary Secretary: Mr. F. C. W. Capps, 45 Lincoln's Inn Fields, London W. C. 2, England

BRITISH MEDICAL ASSOCIATION, SECTION ON OPHTHALMOLOGY

President: Mr. O. G. Morgan
Secretary: Mr. A. G. Cross, 27 Harley Street, London W. 1, England

CENTRO DE ESTUDOS DE OFTALMOLOGIA DE SAO PAULO

President: Dr. Rubens Belfort Mattos
Secretary: Dr. Jose Carlos Gouvea Pacheco, Marconi 34, Sao Paulo

CHENGTU OPHTHALMOLOGICAL SOCIETY

President: Dr. Eugene Chan
Secretary: Dr. D. S. Shen, Eye, Ear, Nose and Throat Hospital, Chengtu, Szechuan, China
Place: Eye, Ear, Nose and Throat Hospital, Chengtu, Szechuan, China

CHINESE OPHTHALMOLOGY SOCIETY

President: Dr. C. H. Chou
Secretary: Dr. F. S. Tsang, 221 Foochow Road, Shanghai

CHINESE OPHTHALMOLOGICAL SOCIETY OF PEKING

President: Dr. G. C. Lin
Secretary: Dr. H. L. Chen, Ophthalmological Dept., Peking University Medical School, Peking
Time: Bimonthly

DEUTSCHE OPHTHALMOLOGISCHE GESELLSCHAFT HEIDELBERG

President: Prof. Dr. med. K. Wessely
Secretary: Prof. Dr. med. E. Engelking, Heidelberg, Universitäts-Augenklinik

FACULTY OF OPHTHALMOLOGISTS

President: Mr. O. M. Duthie
Secretary: Mr. J. H. Doggart, F.R.C.S., 45 Lincoln's Inn Fields, London, W.C. 2, England

FRENCH SOCIETY OF OPHTHALMOLOGY

Secretary: Dr. Edward Hartmann, 2 Ave Ingres, Paris 16, France

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Secretaries of societies are requested to furnish the information necessary to make this list complete and keep it up to date.

GERMAN OPHTHALMOLOGICAL SOCIETY
President: Prof. W. Lohlein
Secretary: Prof. E. Engelking, Heidelberg

HUNGARIAN MEDICAL TRADE UNION, SECTION OF OPHTHALMOLOGY
President: Prof. I. Csapody
Secretary: Dr. E. Galla, Krisztina Körut 139, Budapest, Hungary
Time and Place: Bimonthly. Second Eye Clinic, Budapest

HUNGARIAN OPHTHALMOLOGICAL SOCIETY
President: Prof. G. Horay
Assistant Secretary: Dr. Stephen de Grosz, University Eye Hospital, No. 1, Illesucca 15, Budapest

ISRAEL OPHTHALMOLOGICAL SOCIETY
President: Dr. Aryeh Feigenbaum
Secretary: Dr. E. Sinai, 9 Bialik Street, Tel Aviv

MIDLAND OPHTHALMOLOGICAL SOCIETY
President: Dr. F. A. Anderson
Secretaries: Dr. P. Jameson Evans, 51 Calthorpe Road, Edgbaston, Birmingham 15, England
Dr. R. D. Weeden Butler, 18 Highfield Road, Edgbaston, Birmingham 15, England
Place: Birmingham and Midland Eye Hospital, Church Street, Birmingham 3, England

NEDERLANDSCH OOGHEELKUNDIG GEZELSCHAP
President: Prof. A. W. Mulock Houwer
Secretary: Dr. T. A. Vos, Laan v. Meerdervoort 394, the Hague

NORTH OF ENGLAND OPHTHALMOLOGICAL SOCIETY
President: Mr. A. McKie Reid
Secretary: Mr. W. M. Muirhead, 70 Upper Hanover Street, Sheffield 3, England
Time and Place: October to May. Manchester, Leeds, Newcastle-on-Tyne, Liverpool, Sheffield and Bradford

THE NOVA SCOTIA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. C. K. Fuller
Secretary-Treasurer: Dr. E. I. Glenister, 513 Barrington Street, Halifax, Nova Scotia
Time: Four times yearly at dates to be arranged by the Executive

OPHTHALMOLOGICAL SOCIETY OF AUSTRALIA
President: Dr. G. A. Brew
Secretary: Dr. Arnold L. Lance, 135 Macquarie Street, Sydney, New South Wales
Time and Place: August 1953, in Melbourne

OPHTHALMOLOGICAL SOCIETY OF COPENHAGEN
President: Prof. Dr. Holger Ehlers
Secretary: Dr. Carl Johan Moellenbach, Rigshospitalets oejenafdeling, Copenhagen O, Denmark
Place: Rigshospitalets oejenafdeling

OPHTHALMOLOGICAL SOCIETY OF EGYPT
President: Dr. Ibrahim Ahmad Mohammad
Secretary: Dr. Mahmoud Lutfi, Ophthalmic Hospital, Giza
Time and Place: Annual meeting during March. Summer meeting during August. Dar El Hekmah, 42 Kasr El Ainy Street, Cairo, Egypt

OPHTHALMOLOGICAL SOCIETY OF HOSPITAL DE NUESTRA SENORA DE LA LUZ
Chairman: Dr. Manuel J. Icaza y Dublan
Secretary: Dr. Jorge Meyran, Ezequiel Montes 135, México, D. F.
Time and Place: Second Friday of each month. Hospital de Nuestra Senora de la Luz

OPHTHALMOLOGICAL SOCIETY OF NEW ZEALAND
President: Dr. L. S. Talbot
Secretary: Dr. W. J. Hope-Robertson, Kelvin Chambers, 16 The Terrace, Wellington
Time and Place: Annually. Auckland, Wellington, Christchurch and Dunedin alternately

OPHTHALMOLOGICAL SOCIETY OF SOUTH AFRICA
President: Dr. L. Staz
Secretary-Treasurer: Dr. J. Guillaume Louw, 901 Dumbarton House, Adderley Street, Cape Town
Time: Annually

OPHTHALMOLOGICAL SOCIETY OF THE UNITED KINGDOM
President: Dr. A. MacRae
Honorary Secretaries: Mr. A. G. Leigh and Mr. Harold Ridley, 45 Lincoln's Inn Fields, London

OPHTHALMOLOGISCHE GESELLSCHAFT IN WIEN
President: K. Lindner
Secretary: Ch. Eyb, Wien, IX, Alserstrasse 4

OXFORD OPHTHALMOLOGICAL CONGRESS
Master: Dr. F. A. Anderson
Hon. Secretary and Treasurer: Ian C. Fraser, F.R.C.S., Red Roofs, Kingsland, Shrewsbury

PHILIPPINE OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY
President: Dr. Jesus Eusebio
Secretary-Treasurer: Dr. Jesus Tamesis, Philippine General Hospital, Manila

POLISH OPHTHALMOLOGICAL SOCIETY
President: Prof. Dr. W. Kapuscinski
Secretary: Dr. S. Topolski, Piusa 38, Warsaw, Poland
Time and Place: Every two years—summer. Ophthalmic Clinic, Oczki 6, Warsaw

ROYAL SOCIETY OF MEDICINE, SECTION OF OPHTHALMOLOGY
President: Montague Hine, F.R.C.S.
Secretaries: Arthur Lister, F.R.C.S., 56 Wimpole Street, London, W.1., England
H. E. Hobbs, F.R.C.S., 129 Harley Street, London, W.1., England

SAO PAULO SOCIETY OF OPHTHALMOLOGY
President: Dr. Durval Prado
Secretary: Dr. Rubens Belfort Mattos, Rua B. Stapetininga, 29 F-3° andar, Sao Paulo, Brazil

SCOTTISH OPHTHALMOLOGICAL CLUB
President: Dr. E. H. Cameron
Secretary: Dr. John Marshall, 11 Clairmont Gardens, Glasgow, C.3, Scotland
Time and Place: Last Saturday of March and October, Edinburgh and Glasgow, in rotation

SOCIEDAD ARGENTINA DE OFTALMOLOGIA
President: Dr. Jorge Malbran
Secretary: Dr. Jose Maria Roveda, Lezica 4472, Buenos Aires
Time and Place: Third Wednesday of every month. Buenos Aires

SOCIEDAD CHILENA DE OFTALMOLOGIA
President: Dr. Italo Martini
Secretary: Dr. Adrian Araya Costa, Renaca, no. 34, Santiago, Chile

SOCIEDAD COLOMBIANA DE OFTALMOLOGIA Y DE OTORHINOLARINGOLOGIA
President: Dr. Jorge Suarez-Hoyos, Carrera 5a, no. 13-39, Bogota, Colombia
Secretary: Dr. Francisco Arango
Time and Place: Second Tuesday of each month. Club Medico

SOCIEDAD CUBANA DE OFTALMOLOGIA
President: Dr. Oscar F. Horstmann
Secretary: Dr. Heriberto Buch Granados, Calle B No. 668 ent. 27 y 29, Vedado, Habana, Cuba
Time and Place: First Thursday bi-monthly. Malecon No. 61 Bajos, Havana, Cuba

SOCIEDAD MEXICANA DE OFTALMOLOGIA
President: Dr. Teodulo M. Agundis
Secretary: Dr. Jose Luis Arce, Viena 3-5, Mexico D. F., Mexico
Time and Place: 8:30 p.m., first Tuesday of each month, Gral. PRIM 47

SOCIEDAD NICARAGUIENSE DE OFTALMOLOGIA
President: Dr. Ricardo Lacayo
Secretary: Dr. Gilberto Cuadra, Clinica Especializada, Managua, D. N., Nicaragua, C. A.

SOCIEDAD DE OFTALMOLOGIA DE CORDOBA
Chairman: Dante J. Yadarola
Secretary: Dr. Alberto Urrets Zavalia (hijo), 27 de Abril 255, Cordoba, Argentina

SOCIEDAD DE OFTALMOLOGIA DE GUADALAJARA
President: Dr. Elias Mendoza Gonzalez
Secretary: Dr. Jose Martin del Campo, Av. Juarez 211, Desp. 314, Guadalajara, Jal.
Place: Edificio Lutencia Despacho 101

SOCIEDAD OFTALMOLOGICA HISPANO AMERICANA
President: Dr. Soria
Secretary: Dr. Costi, 3. Calle Montalban, Madrid

SOCIEDAD DE OFTALMOLOGIA DEL LITORAL
President: Dr. Juan Manuel Vila Ortiz, Cordoba 1915, Rosario, Argentina
Secretary: Dr. Carlos M. Soto
Time and Place: Last Sunday in every month. Rosario

SOCIEDAD OFTALMOLOGICA DE VALPARAISO
President: Prof. Dr. Jean H. Thierry
Secretary-Treasurer: Dr. Jorge Oyarzun O, Hospital de Ojos, Calle Blas, Cuevas, Valparaiso

SOCIEDAD PERUANA DE OTO-RINO-LARINGOLOGIA Y OFTALMOLOGIA
President: Dr. Enrique Cipriani V., Pasaje Olaya 156, Lima, Peru
Secretario General: Dr. Enrique Haro
Secretario de Actas: Dr. Alberto Castillo R.

SOCIEDADE BRASILEIRA DE OFTALMOLOGIA
President: Dr. Paulo C. Pimental
First Secretary: Dr. Pedro Moacyr de Aguiar, Ladeira Santa Thereza 136, Apt. 101, Rio de Janeiro
Second Secretary: Dr. Luiz Enrico Ferreira
Time: Third Friday of every month from April to December

SOCIEDADE DE OFTALMOLOGIA DEL NORTE
President: Dr. Jorge Luis Castillo
Secretary: Dr. Felix Berman, San Lorenzo 345, Tucuman, Argentina
Place: Mendoza 421, Tucuman

SOCIEDADE DE OFTALMOLOGIA DE MINAS GERAES
President: Dr. Amelio Bonfioli
Secretary: Dr. Mariana Noronha, Pauso Alegre 1150, Belo Horizonte, Est. Minas Geraes

SOCIEDADE DE OFTALMOLOGIA E OTO-RHINO-LARYNGOLOGIA DE BAHIA
President: Dr. Theonilo Amorim, Barra Avenida, Bahia, Brazil
Secretary: Dr. Adroaldo de Alencar

SOCIEDADE DE OFTALMOLOGIA E OTO-RINO-LARINGOLOGIA DO RIO GRANDE DO SUL
President: Dr. Mario Araujo Azambuja
First Secretary: Dr. Humberto Lubisco, Rua Donna Laura 45, Porto Alegre, Est., Rio Grande do Sul
Second Secretary: Dr. Ivo Kuhl

SOCIEDADE DE OFTALMOLOGIA DE SAO PAULO
President: Dr. Paulo Braga Magalhaes
General Secretary: Dr. Paulo Aranha de Azevedo, Xavier de Toledo 71,1° and., Sao Paulo
First Secretary: Dr. Alfredo Rocco

SOCIEDADE DE OTO-RINO-LARINGOLOGIA DO RIO DE JANIERO
President: Dr. Aloysio Novis
Secretaries: Dr. Rubens Cabral, Rua Paissandu 73, Rio de Janeiro, Brazil
Dr. Mairelles Vieira

SOCIETA OFTALMOLOGICA ITALIANA

President: Prof. Giuseppe Ovio
Secretary: Prof. E. Leonardi, Piazza degli Eroi, 11 Roma, Italy
Place: Roma Piazza degli Eroi 11

SOCIETE BELGE D'OPHTALMOLOGIE

President: Dr. L. Weekers
Secretary-General: Dr. M. Appelmans, 179 avenue Reulens, Louvain, Belgium
Time: Last Sunday of February, June and November

SOCIETE D'OTO-RHINO-LARYNGOLOGIE

Administrateur: G. Raimond, 17et19, Rue de Buci, Paris (VI.) France

SOCIETY OF SWEDISH OPHTHALMOLOGISTS

President: Prof. S. Larsson
Secretary: Dr. K. O. Granstrom, Sodermalmstorg 4, Stockholm, Sweden

SOUTHERN OPHTHALMOLOGICAL SOCIETY, ENGLAND

President: Campbell Shaw, M. R. C. S.
Hon. Secretary and Treasurer: Nigel Cridland, D.M., D.O., 25 Craneswater Park, Southsea, England

NATIONAL

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. James Milton Robb
Executive Secretary-Treasurer: Dr. William L. Benedict, 100 First Avenue Building, Rochester, Minn.
Time and Place: October 12-17, 1952, Chicago

AMERICAN ASSOCIATION OF EYE, EAR, NOSE AND THROAT SOCIETY SECRETARIES

President: Dr. M. E. Alvaro
Secretary-Treasurer: Dr. Daniel S. DeStio, Highland Building, Pittsburgh 6, Pa.

AMERICAN LARYNGOLOGICAL ASSOCIATION

President: Dr. Gordon B. New
Secretary: Dr. Louis H. Clerf, 1530 Locust, Philadelphia 2, Pa.

AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY, INC.

President: Dr. C. Stewart Nash
Secretary: Dr. C. Stewart Nash, 708 Medical Arts Building, Rochester 7, N. Y.

EASTERN SECTION
Vice-President: Dr. Francis W. Davison
Secretary: Dr. Francis W. Davison, Geisinger Memorial Hospital, Danville, Pa.

MIDDLE SECTION
Vice-President: Dr. Ralph J. McQuiston
Secretary: Dr. Walter H. Theobald, 307 North Michigan Avenue, Chicago 1, Ill.

SOUTHERN SECTION:
Vice-President: Dr. Lester A. Brown
Secretary: Dr. James W. McLaurin, Raymond Building, Baton Rouge 6, La.

WESTERN SECTION:
Vice-President: Dr. Howard P. House
Secretary: Dr. Harold Boyd, 1136 West Sixth Street, Los Angeles, Calif.

AMERICAN MEDICAL ASSOCIATION, SCIENTIFIC ASSEMBLY, SECTION ON OPHTHALMOLOGY

Chairman: Dr. A. Ray Irvine
Secretary: Dr. Trygve Gundersen, 101 Bay State Road, Boston, Mass.

AMERICAN MEDICAL ASSOCIATION, SECTION ON LARYNGOLOGY, OTOLOGY AND RHINOLOGY

Chairman: Dr. James Milton Robb
Secretary: Dr. Sam H. Sanders, 1089 Madison Avenue, Memphis 3, Tenn.

AMERICAN OPHTHALMOLOGICAL SOCIETY

President: Dr. Lawrence T. Post
Secretary-Treasurer: Dr. Maynard C. Wheeler, 30 W. 59th Street, New York 19, N. Y.

AMERICAN OTOLOGICAL SOCIETY

President: Dr. Kenneth Day
Secretary: Dr. John R. Lindsay, 950 East 59th Street, Chicago 37, Ill.

AMERICAN OTORHINOLOGIC SOCIETY FOR THE ADVANCEMENT OF PLASTIC AND RECONSTRUCTIVE SURGERY

President: Dr. Norman N. Smith
Secretary: Dr. Joseph G. Gilbert, 1404 President Street, Brooklyn 13, N. Y.

AMERICAN SOCIETY OF OPHTHALMOLOGIC AND OTOLARYNGOLOGIC ALLERGY

President: Dr. William H. Evans
Secretary-Treasurer: Dr. Joseph W. Hampsey, 806 May Building, Pittsburgh 22, Pa.
Time and Place: Annual meeting, October 17, 1952, Chicago

ASSOCIATION FOR RESEARCH IN OPHTHALMOLOGY, INC.

Chairman: Dr. Walter H. Fink
Secretary-Treasurer: Dr. James H. Allen, 1430 Tulane Avenue, New Orleans, La.

CANADIAN MEDICAL ASSOCIATION, SECTION ON OPHTHALMOLOGY

President: Dr. R. G. C. Kelly
Secretary: Dr. J. Clement McCulloch, 380 Medical Arts Building, Toronto, Ont.

CANADIAN OPHTHALMOLOGICAL SOCIETY

President: Dr. R. F. Nicholls
Secretary. Dr. J. F. A. Johnston, 174 St. George Street, Toronto, Ont.

CANADIAN OTOLARYNGOLOGICAL SOCIETY

President: Dr. Jules Brahy
Secretary: Dr. W. Ross Wright, 361 Regent Street, Fredericton, New Brunswick

NATIONAL SOCIETY FOR THE PREVENTION OF BLINDNESS, INC.
President: Mr. Mason H. Bigelow
Secretary: Dr. Franklin M. Foote, 1790 Broadway, New York 19, N. Y.

REGIONAL

ARK-LA-TEX OTO-OPHTHALMIC SOCIETY
President: Dr. Joel A. Wilkinson
Secretary: Dr. Frank L. Bryant, 2622 Greenwood, Shreveport, La.
Time and Place: First Monday of month, September through June. Shreveport Club

HAWAII EYE, EAR, NOSE AND THROAT SOCIETY
Chairman: Dr. John P. Frazer
Secretary: Dr. C. W. Trexler, Young Hotel Building, Honolulu, T. H.
Time and Place: Each Thursday (dinner meetings) 6:30 p.m., Pacific Club, Honolulu

INTER-MOUNTAIN OTO-OPHTHALMOLOGICAL SOCIETY
President: Dr. Harry O. Frazier
Secretary: Dr. Robert L. Rees, 115 East South Temple Street, Salt Lake City, Utah
Time and Place: Third Monday of each month, September through May. University Club, 136 East South Temple, Salt Lake City

LOUISIANA-MISSISSIPPI OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY
President: Dr. William B. Clark
Secretary: Dr. Edley H. Jones, 1301 Washington Street, Vicksburg, Miss.

NASSAU OPHTHALMOLOGICAL SOCIETY
President: Dr. A. Milton Goldman
Secretary: Dr. Ward L. Mould, 131 Fulton Avenue, Hempstead, N. Y.
Time and Place: Fourth Monday in February, April, September and November at 7:30 p.m. at Meadowbrook Hospital, Hempstead, N. Y.

NEW ENGLAND OPHTHALMOLOGICAL SOCIETY
President: Dr. Benjamin A. Sachs
Secretary: Dr. Garrett L. Sullivan, 101 Bay State Road, Boston 15, Mass.
Time and Place: Third Wednesday of each month, November through April. Massachusetts Eye and Ear Infirmary

NEW ENGLAND OTO-LARYNGOLOGICAL SOCIETY
President: Dr. Leighton F. Johnson
Secretary-Treasurer: Dr. Burton E. Lovesey, 76 Bay State Road, Boston 15, Mass.
Time and Place: Quarterly. Massachusetts Eye and Ear Infirmary, 243 Charles Street, Boston

PACIFIC COAST OTO-OPHTHALMOLOGICAL SOCIETY
President: Dr. C. Allen Dickey
Secretary-Treasurer: Dr. Howard P. House, 1136 West Sixth Street, Los Angeles 14, Calif.
Time and Place: 1953, Los Angeles, Calif.

SAGINAW VALLEY ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. William B. Hubbard
Secretary: Dr. Frank A. Ware, 514 Genesee Bank Bldg., Flint, Mich.
Time and Place: Second Tuesday evening of each month, except summer months. Bancroft Hotel, Saginaw

SIOUX VALLEY EYE AND EAR ACADEMY
President: Dr. James Reeder, Jr.
Secretary-Treasurer: Dr. W. P. Davey, 627-632 Frances Building, Sioux City, Iowa

SOUTHERN MEDICAL ASSOCIATION, SECTION ON OPHTHALMOLOGY AND OTOLARYNGOLOGY
Chairman: Dr. Edley H. Jones
Secretary: Dr. F. A. Holden, Medical Arts Building, Baltimore, Md.
Time and Place: November 10-13, 1952, Miami, Fla.

TIDEWATER OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY
President: Dr. C. C. Cooley
Secretary: Dr. J. G. Sellers, 413 Medical Arts Building, Norfolk, Va.

WISCONSIN-UPPER MICHIGAN SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. James K. Trumbo
Secretary: Dr. G. L. McCormick, 650 South Central Avenue, Marshfield, Wis.

STATE

ALABAMA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. Eugene Calloway, Sr.
Secretary-Treasurer: Dr. Karl B. Benkwith, Catoma Street, Montgomery 5, Ala.

ARKANSAS STATE MEDICAL SOCIETY, EYE, EAR, NOSE AND THROAT SECTION
President: Dr. C. G. Hinkle
Secretary: Dr. K. W. Cosgrove, 113 East Capitol Avenue, Little Rock, Ark.

CALIFORNIA MEDICAL ASSOCIATION, EYE, EAR, NOSE AND THROAT SECTION
Chairman: Dr. George F. Keiper, Jr.
Secretary: Dr. Maurice W. Nugent, 2007 Wilshire Blvd., Los Angeles 5, Calif.

COLORADO OPHTHALMOLOGICAL SOCIETY
President: Dr. J. Leonard Swigert
Secretary: Dr. James C. Strong, 227 Sixteenth Street, Denver, Colo.
Time and Place: Every third Saturday. Colorado Medical Center

COLORADO OTOLARYNGOLOGICAL SOCIETY
President: Dr. Ivan E. Hix
Secretary: Dr. Merrill O. Dart, 3191 S. Broadway, Englewood, Colo.
Time and Place: First Saturday of each month, October through May. Colorado University School of Medicine

FLORIDA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. Charles C. Grace
Secretary-Treasurer: Dr. Carl S. McLemore, 1202 South Kuhl Avenue, Orlando, Fla.

GEORGIA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. Stacy C. Howell
Secretary: Dr. Alton V. Hallum, 245 Doctors Building, Augusta 3, Ga.

INDIANA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. E. L. Van Buskirk
Secretary-Treasurer: Dr. John R. Swan, 23 East Ohio Street, Indianapolis 4, Ind.

IOWA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. Byron Merkel
Secretary: Dr. Carl A. Noe, 601-611 Highley Building, Cedar Rapids, Iowa

KANSAS STATE MEDICAL SOCIETY, SECTION ON OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. N. L. Francis
Secretary: Dr. W. D. Pitman, First National Bank Building, Pratt, Kan.

MASSACHUSETTS MEDICAL SOCIETY, SECTION ON OPHTHALMOLOGY AND OTOLARYNGOLOGY
Chairman: Dr. Lyman Richards
Secretary: Dr. Lawrence R. Dame, 78 Federal Street, Greenfield, Mass.

MICHIGAN STATE MEDICAL SOCIETY, SECTION ON OPHTHALMOLOGY AND OTOLARYNGOLOGY
Chairman: Dr. J. E. Croushore (Otolaryngology)
Co-Chairman: Dr. J. C. Gemeroy (Ophthalmology)
Secretary: Dr. R. W. Teed, 215 South Main, Ann Arbor, Mich. (Otolaryngology)
Co-Secretary: Dr. F. B. Heckert, 1105 Bank of Lansing Building, Lansing, Mich. (Ophthalmology)

MICHIGAN TRIOLOGICAL SOCIETY
President: Dr. William D. Irwin
Secretary: Dr. V. E. Cortopassi, 324 South Washington Avenue, Saginaw, Mich.
Time: Second Thursday of November, December, March, April and May

MINNESOTA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. K. A. Phelps
Secretary-Treasurer: Dr. R. W Holmen, Lowry Medical Arts Building, St. Paul 2, Minn.
Time and Place: Second Friday of each month, November through May. Alternating, Minnesota Club, St. Paul, and Minneapolis Club, Minneapolis

MONTANA ACADEMY OF OTO-OPHTHALMOLOGY
President: Dr. Fritz D. Hurd
Secretary-Treasurer: Dr. H. L. Casebeer, Pennsylvania Building, 44 West Park Street, Butte, Mont.
Time and Place: September 19, 1952, Missoula, Mont.

NEW MEXICO OPHTHALMOLOGIC SOCIETY
President: Dr. A. L. Schonberg
Secretary: Dr Howard Peck, Medical Arts Square, Albuquerque, N. M.
Time: Meetings four times a year

NORTH CAROLINA EYE, EAR, NOSE AND THROAT SOCIETY
President: Dr. MacLean B. Leath
Secretary-Treasurer: Dr. George B. Ferguson, 1110 West Main St., Durham, N. C.
Time and Place: September 15-17, 1952, Hotel Robert E. Lee, Winston-Salem, N. C.

OREGON ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. Richard S. Fixott
Secretary: Dr. George E. Chamberlain, 909 Medical Dental Building, Portland 5, Ore.
Time and Place: Third Tuesday each month September through May. Old Heathman Hotel, Portland

PENNSYLVANIA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. Matthew S. Ersner
Secretary: Dr. Daniel S. DeStio, Highland Building, Pittsburgh 6, Pa.

PUERTO RICO MEDICAL ASSOCIATION, SECTION ON OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. Luis J. Fernandez
Secretaries: Dr. Ricardo F. Fernandez, P.O. Box 2206, San Juan 10, Puerto Rico (Ophthalmology)
Dr. Carlos E. Munoz MacCormick, P.O. Box 604, San Juan, Puerto Rico (Otolaryngology)
Time and Place: Bimonthly. Puerto Rico Medical Association Building

SOUTH CAROLINA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. Thomas R. Gaines
Secretary-Treasurer: Dr. Roderick Macdonald, 330 East Main Street, Rock Hill, S. C.
Time and Place: September 15-17, 1952, Hotel Robert E. Lee, Winston-Salem, N. C.

TENNESSEE STATE ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. Roland H. Myers
Secretary-Treasurer: Dr. John T. Bryan, 2105 Hayes Street, Nashville, Tenn.
Time: Annually

TEXAS SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. J. Charles Dickson
Secretary: Dr. Lyle Hooker, Esperson Building, Houston, Texas

VIRGINIA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. A. D. Morgan
Secretary-Treasurer: Dr. G. Slaughter Fitz-Hugh, 104 East Market St., Charlottesville, Va.
Time and Place: Fall Clinical Session: Ophthalmology, November 18-19, 1952; Otolaryngology, November 20-21, 1952, Charlottesville, Va.; Spring meeting, May 1953, Hot Springs, Va.

WEST VIRGINIA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. Arthur Chandler
Secretary: Dr. Ben W. Bird, 909 Mercer St., Princeton, W. Va.
Time: Biannual

LOCAL

ACADEMY OF MEDICINE OF NORTHERN NEW JERSEY, SECTION ON EYE, EAR, NOSE AND THROAT
Chairman: Dr. William F. Krone
Secretary: Dr. Francis J. Grant, 1224 Salem Avenue, Hillside, N. J.
Time and Place: 8:45 p.m., second Monday of the month. The Academy of Medicine, 91 Lincoln Park, South, Newark

AKRON ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. C. R. Anderson
Secretary-Treasurer: Dr. A. L. Peter, 406 Akron Savings and Loan Building, Akron, Ohio
Time and Place: 6:30 p.m., first Monday in January March, May and November. Akron City Club

ALUMNI ASSOCIATION NEW YORK EYE AND EAR INFIRMARY

President: Dr. W. T. Boland
Secretary-Treasurer: Dr. Hunter Romaine, 111 East 65th Street, New York, N. Y.

ATLANTA EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. F. Phinizy Calhoun, Jr.
Secretary-Treasurer: Dr. James T. King, 384 Peachtree Street N.E., Atlanta 3, Ga.
Time and Place: 7:30 p.m., fourth Monday of each month, October to May. Academy of Medicine

BALTIMORE CITY MEDICAL SOCIETY, OPHTHALMOLOGICAL SECTION

Chairman: Dr. M. Elliott Randolph
Secretary-Treasurer: Dr. Abraham Kremen, 2355 Eutaw Place, Baltimore 17, Md.
Time and Place: 8:00 p.m., fourth Thursday of every month October to May. Medical and Chirurgical Faculty, 1211 Cathedral St.

BIRMINGHAM EYE, EAR, NOSE AND THROAT SOCIETY

President: Each member, in alphabetical order.
Secretary: Dr. David A. McCoy, Woodward Building, Birmingham, Ala.
Time and Place: 6:30 p.m., second Tuesday of each month, September through May. Thomas Jefferson Hotel

BOSTON CITY HOSPITAL AURAL AND OPHTHALMIC ASSOCIATION

President: Dr. Joseph Nerbonne
Secretary: Dr. Benjamin Riseman, 41 Bay State Road, Boston 15, Mass.

BROOKLYN OPHTHALMOLOGICAL SOCIETY

President: Dr. Daniel K. Kravitz
Secretary-Treasurer: Dr. Louis Freimark, 256 Rochester Avenue, Brooklyn 13, N. Y.
Time and Place: 8 p.m., third Thursday in October, December, February and April. Medical Society of the County of Kings, 1313 Bedford Avenue

BUFFALO OPHTHALMOLOGIC CLUB

President: Dr. Milton A. Palmer
Secretary: Dr. Elizabeth P. Olmsted, 568 Lafayette Avenue, Buffalo 22, N. Y.
Time and Place: Second Thursday of each month, October through May. Park Lane

CENTRAL ILLINOIS SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. G. C. Otrich
Secretary-Treasurer: Dr. Alfred G. Schultz, 237 East State Street, Jacksonville, Ill.

CENTRAL NEW YORK EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. Cecil B. Hert
Secretary-Treasurer: Dr. James L. McGraw, 619 University Building, Syracuse, N. Y.

CHATTANOOGA EYE, EAR, NOSE AND THROAT SOCIETY

Chairman and Secretary: Dr. Willard H. Steele, Jr., 552 McCallie Avenue, Chattanooga, Tenn.
Time and Place: 6:30 p.m., fourth Thursday in each month, October through May. Mountain City Club

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY

President: Dr. Arthur J. Coombs
Secretary: Dr. Lawrence J. Lawson, 636 Church Street, Evanston, Ill.

CHICAGO OPHTHALMOLOGICAL SOCIETY

President: Dr. William F. Hughes, Jr.
Secretary-Treasurer: Dr. Gail R. Soper, 636 Church Street, Evanston, Ill.
Time and Place: 7:30 p.m., third Monday of each month, October to May. Chicago-Illini Union, 715 S. Wood Street

CINCINNATI OPHTHALMOLOGIC CLUB

Presidium: Dr. Donald J. Lyle, Dr. Karl W. Ascher, Dr. Josef D. Weintraub
Secretary: Dr. Josef D. Weintraub, 715 Provident Bank Building, Seventh and Vine Streets, Cincinnati, Ohio
Time and Place: Second Wednesday night of month, November through May. University Club, Fourth and Broadway

CINCINNATI OTOLARYNGOLOGIC SOCIETY

President: Dr. Raymond L. Hilsinger
Secretary-Treasurer: Dr. Stephen P. Hogg, 556 Doctors' Building, Cincinnati 2, Ohio
Time and Place: 6:30 on the third Monday of each month except July, August, September and December. University Club.

CLEVELAND OPHTHALMOLOGICAL CLUB

President: Dr. G. Leslie Miller
Secretary-Treasurer: Dr. Webb P. Chamberlain, Jr., 7405 Detroit Avenue, Cleveland, Ohio
Time and Place: Second Tuesday of November, January, February and April. Statler Hotel, Cleveland

COLLEGE OF PHYSICIANS OF PHILADELPHIA, SECTION ON OPHTHALMOLOGY

Chairman: Dr. George F. J. Kelly
Clerk: Dr. M. Luther Kauffman, Medical Arts Building, Jenkintown, Pa.
Time and Place: 8:15 p.m., third Thursday of every month, October through April. College of Physicians Building

CORPUS CHRISTI EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. Rex E. House
Secretary-Treasurer: Dr. L. David Wright, Thomas-Spann Clinic, 1546 South Brownlee, Corpus Christi, Texas
Time and Place: First Wednesday of each month. La Louisiane

DALLAS ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. W. B. Wilkinson
Secretary: Dr. Claude D. Winborn, Medical Arts Building, Dallas 1, Texas
Time and Place: First Tuesday of each month. Melrose Hotel

DES MOINES ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. J. H. McNamee
Secretary-Treasurer: Dr. H. H. Gurau, 213 Bankers Trust Building, Des Moines, Iowa
Time and Place: 6:00 p.m., fourth Monday of each month, September through May. Des Moines Club

DETROIT OPHTHALMOLOGICAL CLUB

Secretary-Treasurer: Dr. Frederick A. Lauppe, 1249 David Whitney Building, Detroit 26, Mich.

DETROIT OPHTHALMOLOGICAL SOCIETY

President: Dr. Arthur S. Hale
Secretary: Dr. Arthur P. Wilkinson, 974 Fisher Building, Detroit 2, Mich.
Time and Place: Fourth Thursday of each month, November through April, at L'Aiglon, Fisher Building

DETROIT OTO-LARYNGOLOGICAL SOCIETY
President: Dr. B. F. Glowacki
Secretary-Treasurer: Dr. John R. Birch, 1010 Maccabees Building, Detroit 2, Mich.
Time and Place: Third Wednesday of each month, September to May. Wayne County Medical Society Building

EASTERN NEW YORK EYE, EAR, NOSE AND THROAT ASSOCIATION
President: Dr. Harry Miller
Secretary-Treasurer: Dr. Frank Furlong, 713 Union Street, Schenectady, N. Y.
Time and Place: 8 p.m., first Thursday of month, October to June. Albany, Troy, Schenectady, rotating monthly

FORT WORTH EYE, EAR, NOSE AND THROAT SOCIETY
President: Dr. Hal Maxwell
Secretary: Dr. William Skokan, 1708 Medical Arts Building, Fort Worth 2, Texas
Time and Place: 6:30 p.m., first Friday of each month, except July and August. St. Joseph's Hospital

HOUSTON ACADEMY OF MEDICINE, OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SECTION
President: Dr. Lyle Hooker
Secretary: Dr. Claude C. Cody, III, 1304 Walker Avenue, Houston 2, Texas
Time: Second Thursday of each month, October through June

INDIANAPOLIS OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SOCIETY
President: Dr. Marlow W. Manion
Secretary-Treasurer: Dr. Lewis E. Morrison, 603 Hume Mansur Building, Indianapolis 4, Ind.
Time and Place: 6:30 p.m., second Thursday of each month, November to May. Indianapolis Athletic Club

KANSAS CITY SOCIETY OF O. O. R. L.
President: Dr. A. W. McAlester, III
Secretary: Dr. Barnard C. Trowbridge, 411 Alameda Road, Kansas City 2, Mo.
Time and Place: Third Thursday of the month, November through May. Hotel President, 14th and Baltimore

LONG BEACH EYE, EAR, NOSE AND THROAT SOCIETY
President: Dr. Edmund D. Godwin
Secretary: Dr. James V. Keipp, 110 Pine Avenue, Long Beach 2, Calif.
Time and Place: 6:00 p.m., monthly, September through May. Seaside Memorial Hospital

LOS ANGELES OPHTHALMOLOGICAL SOCIETY
President: Dr. Deane C. Hartman
Secretary: Dr. Daniel B. Esterly, 104 North Madison Avenue, Pasadena 1, Calif.
Time and Place: First Thursday of each month, September through June. Los Angeles County Medical Society Building, 1925 Wilshire Boulevard

LOS ANGELES SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. Victor Goodhill
Secretary: Dr. Orwyn Ellis, 523 West Sixth Street, Los Angeles 14, Calif.
Time and Place: 6:00 p.m., fourth Monday of each month, September through May. Los Angeles County Medical Association Building, 1925 Wilshire Boulevard

MEDICAL SOCIETY OF THE DISTRICT OF COLUMBIA, SECTION ON OPHTHALMOLOGY
President: Dr. Everett S. Caldemeyer
Secretary-Treasurer: Dr. J. Spencer Dryden, 1835 Eye Street Northwest, Washington 6, D. C.

MEDICAL SOCIETY OF THE DISTRICT OF COLUMBIA, SECTION ON OTOLARYNGOLOGY
Chairman: Dr. Irvin Feldman
Secretary: Dr. John Louzan, 1801 Eye Street N. W., Washington 6, D. C.
Time and Place: 7:00 p.m., third Tuesday in October, November, January, March and May. Army and Navy Club, 17th and Farragut Square, N.W.

MEMPHIS SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
Chairman: Each member, in alphabetical order
Secretary: Dr. Roland H. Myers, 1720 Exchange Building, Memphis, Tenn.
Time and Place: Second Tuesday in each month, September through May. Memphis Eye, Ear, Nose and Throat Hospital

MILWAUKEE OTO-OPHTHALMIC SOCIETY
President: Dr. J. P. Wild
Secretary-Treasurer: Dr. Howard High, 324 East Wisconsin Avenue, Milwaukee 2, Wis.
Time and Place: 6:30 p.m., fourth Tuesday of each month, October to May. Athletic Club

MONTGOMERY COUNTY MEDICAL SOCIETY, SECTION ON EYE, EAR, NOSE AND THROAT
President: Dr. Robert A. Bruce
Secretary-Treasurer: Dr. L. N. Shroder, 144 West Fourth Street, Greenville, Ohio
Time and Place: First Tuesday of November, February, March and May. Dayton Country Club

MONTREAL OPHTHALMOLOGICAL SOCIETY
President: Dr. Jules Brahy
Secretary-Treasurer: Dr. Leo S. S. Kirschberg, 1390 Sherbrooke Street West, Montreal, Que., Canada
Time: Second Thursday of October, December, February and April

NASHVILLE ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. John W. Harned, Jr.
Secretary-Treasurer: Dr. Herbert Duncan, Bennie Dillon Building, Nashville, Tenn.
Time and Place: Third Monday evening of month, October through May. James Robertson Hotel

NEW YORK ACADEMY OF MEDICINE, SECTION ON OPHTHALMOLOGY
Chairman: Dr. James W. Smith
Secretary: Dr. A. Gerard De Voe, 30 West 59th St., New York, N. Y.
Time and Place: Third Monday of the month, October through May. New York Academy of Medicine

NEW YORK SOCIETY FOR CLINICAL OPHTHALMOLOGY
President: Dr. Abraham L. Kornzweig
Secretary: Dr. Leon H. Ehrlich, 211 Central Park West, New York 24, N. Y.
Time and Place: First Monday of the month, October to May. New York Academy of Medicine

NUECES COUNTY EYE, EAR, NOSE AND THROAT SOCIETY
President: Dr. C. N. Meador
Secretary: Dr. Rex C. House, 228 Medical-Dental Building, Corpus Christi, Texas
Time and Place: 7:30 p.m., second Monday of each month, October through May. Dusty's, 1739 South Brownlee Street, Corpus Christi, Texas

OKLAHOMA CITY ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. C. W. McClure
Secretary: Dr. Richard A. Clay, 416 Northwest 13th Street, Oklahoma City, Okla.
Time and Place: First Tuesday of each month. University Hospital, 800 Northeast 13th Street, Oklahoma City

OMAHA AND COUNCIL BLUFFS OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY
President: Dr. Thomas T. Smith
Secretary-Treasurer: Dr. G. T. Alliband, 1020 Medical Arts Building, Omaha, Neb.
Time and Place: Dinner meeting third Wednesday monthly, October to May. Omaha Club

PHILADELPHIA COUNTY MEDICAL SOCIETY, EYE SECTION
Chairman: Dr. Alfred Cowan
Secretary: Dr. John W. Deichler, 301 South 21st Street, Philadelphia 3, Pa.
Time: First Thursday of each month, November through April

PHILADELPHIA LARYNGOLOGICAL SOCIETY
President: Dr. Thomas F. Furlong, Jr.
Secretary: Dr. John J. O'Keefe, 255 South 17th Street, Philadelphia 3, Pa.

PITTSBURGH ACADEMY OF MEDICINE
President: Dr. John S. Plumer
Secretary: Dr. Samuel D. Evans, 1501-2 Park Building, Pittsburgh 22, Pa.
Time and Place: 8:00 p.m., fourth Monday of each month, October through May. Pittsburgh Academy, 322 North Craig Street

PITTSBURGH OTOLOGICAL SOCIETY
President: Dr. Daniel S. DeStio
Secretary: Dr. George C. Schein, 634 Washington Road, Pittsburgh, Pa.
Time and Place: Bimonthly

PUGET SOUND ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY
President: Dr. H. Frederick Thorlakson
Secretary-Treasurer: Dr. Willard F. Goff, 432 Stimson Building, 1215 Fourth Avenue, Seattle 1, Wash.
Time and Place: Third Tuesday of each month. Medical-Dental Building, Seattle

READING EYE, EAR, NOSE AND THROAT SOCIETY
President: Dr. J. Van Dyke Quereau
Secretary: Dr. Benjamin F. Souders, 424 Walnut Street, Reading, Pa.
Time and Place: Third Wednesday of each month, September to July (dinner meetings). Wyomissing Club

RICHMOND, VIRGINIA, EYE, EAR, NOSE AND THROAT SOCIETY
President: Dr. Mason Smith
Secretary-Treasurer: Dr. Charles N. Romaine, 1001 West Franklin Street, Richmond 20, Va.
Time and Place: First Tuesday of January, March, May and October. Commonwealth Club

ST. LOUIS COUNTY MEDICAL SOCIETY, SECTION ON EYE, EAR, NOSE AND THROAT
Chairman: Dr. Anderson C. Hilding
Secretary: Dr. James P. Tetlie, 626 Medical Arts Building, Duluth, Minn.
Time and Place: 6:00 to 8:00 p.m., preceding the monthly St. Louis County Medical Society meeting. St. Mary's Hospital, Duluth

EAR, NOSE AND THROAT CLUB OF ST. LOUIS
Chairman: Dr. Millard F. Arbuckle
Secretary: Dr. Harry N. Glick, 1504 South Grand Boulevard, St. Louis 4, Mo.
Time and Place: Third Wednesday in November, January, March and May, University Club Building

ST. LOUIS OPHTHALMIC SOCIETY
President: Dr. J. M. Keller
Secretary: Dr. Benjamin Milder, 539 North Grand Avenue, St. Louis 3, Mo.
Time and Place: 8:00 p.m., fourth Friday of each month, October through April, except December. Elliott Auditorium, McMillan Hospital

SAN FRANCISCO COUNTY MEDICAL SOCIETY, SECTION ON EYE, EAR, NOSE AND THROAT
Chairman: Dr. W. E. Borley, 655 Sutter Street, San Francisco, Calif.
Secretary: None
Time and Place: Fourth Tuesday of each month, except July, August and December. San Francisco County Medical Society Building

SPOKANE ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. Phil Greene
Secretary: Dr. Cornelius E. Hagan, Jr., 508 Old National Bank Building, Spokane, Wash.
Time and Place: Fourth Tuesday of each month, September through May. Paulsen Medical and Dental Building

SUPERIOR CALIFORNIA EYE CLUB
President: Dr. Theodore Holstein, 523 Medico-Dental Building, Sacramento 14, Calif.
Secretary: Dr. John Berg
Time: Every third Thursday, eight months of the year

TOLEDO EYE, EAR, NOSE AND THROAT SOCIETY
President: Dr. N. Gardner Mathieson
Secretary: Dr. Robert D. Kiess, 1838 Parkwood, Toledo, Ohio
Time and Place: Third Tuesday of each month, November through May, Toledo Club

TORONTO ACADEMY OF MEDICINE, SECTION OF OPHTHALMOLOGY
Chairman: Dr. Joseph C. Hill
Secretary: Dr. Harold R. Sniderman, 220 Bloor Street West, Toronto 5, Ont., Canada
Time and Place: Second Monday night of November, January, February and March. Academy of Medicine, Toronto

WESTERN PENNSYLVANIA EYE, EAR, NOSE AND THROAT SOCIETY
President: Dr. Ralph T. Himes
Secretary-Treasurer: Dr. F. E. Murdock, 28½ West Scribner Avenue, DuBois, Pa.
Time: Biannually. Third Thursdays of May and October

WILKES-BARRE OPHTHALMOLOGICAL SOCIETY
President: Dr. Vincent Gallizzi
Secretary: Dr. Samuel T. Buckman, 70 South Franklin Street, Wilkes-Barre, Pa.
Time and Place: Last Tuesday of each month, October through May, except December. Luzerne County Medical Society Library, 130 South Franklin Street

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Arch. Otolaryngol., 43:605, 1946.
E., E., N., & T. Mo., 26:27, 1947.
Laryngoscope, 56:556, 1946.
New Eng. J. Med., 234:468, 1946.
Annals of Allergy, 4:33, 1946.
J. A. Ph., A., (Sc. Ed.) 35:304, 1946.

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